Industrial Medicine

for

Physicians and Surgeons in Industry

The Pneumonokonioses (Silicosis)

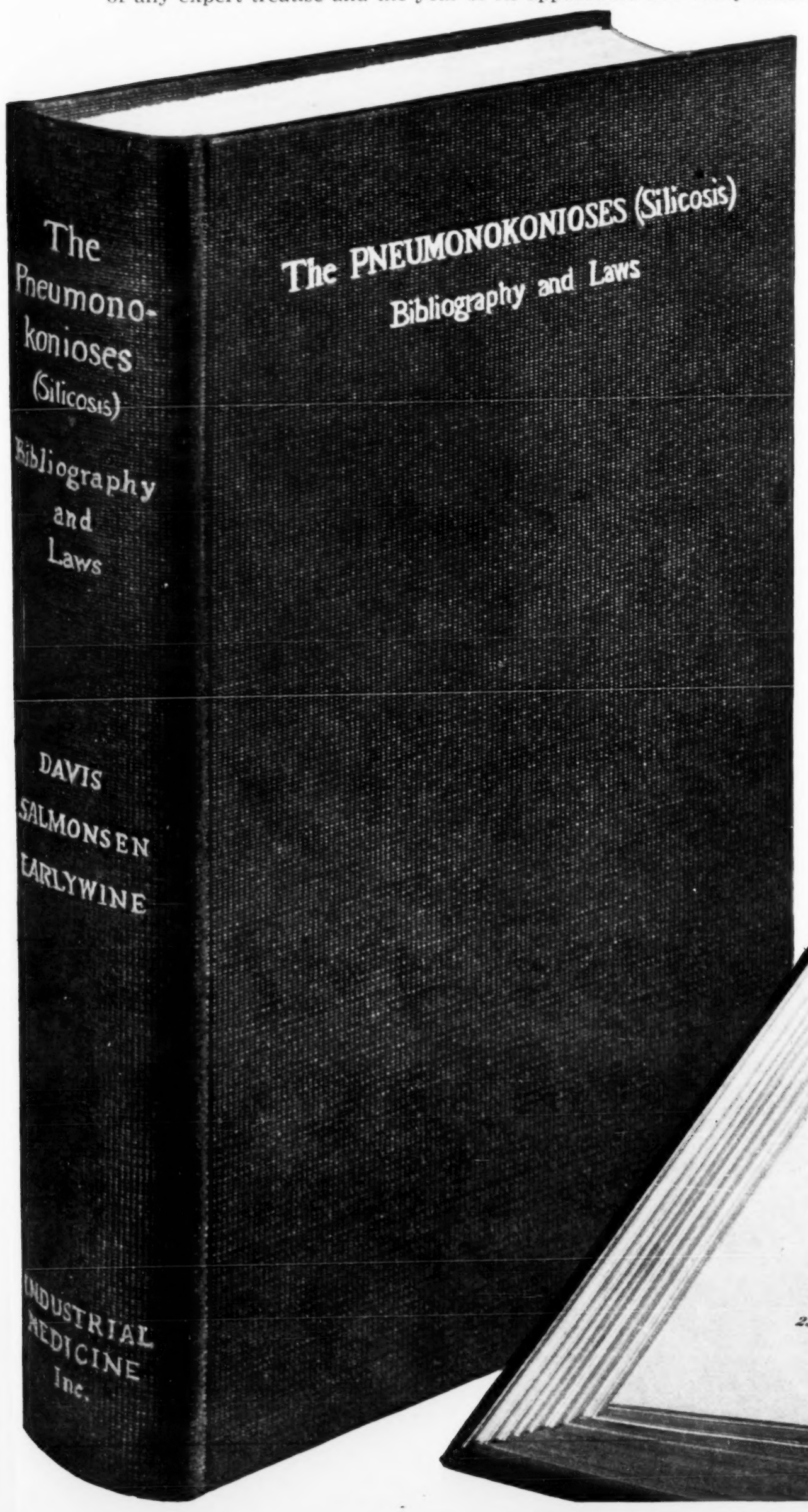
Bibliography *and* Laws Book No. I

All the References of the World's Literature
--- from 1556 to December 31, 1933 ---

Review from **"Staub"**, (Dust), 1, No. 1:167-168, April, 1936, done by Dr. Ing. O. M. Faber, Berlin.

No. 115. Bibliography and Laws upon Silicosis. **(The Pneumonoconioses (Silicosis) Bibliography and Laws),** by Geo. G. Davis, E. M. Salmonsen and J. L. Earlywine, published by **Industrial Medicine,** Chicago, Ill., 1934, 482 pp. ($7.50).

The **Forword** is by E. R. Le Count, Chief of the Department of Pathology, The University of Chicago. The book has two main divisions. Part I is a Bibliography of the collected literature of the world upon the **Pneumonoconioses (Silicosis).** Beginning with the **De Re Metallica** of Agricola (1556), it ends with 1933, inclusive of 2,768 sources, whose title, author and exact citation are given. The works are chronologically arranged. Through subdivision into different groups, the works of authors appearing in the same year are clearly and immediately perceived. Within the groups the works are still arranged in alphabetical sequence by authors. These 276 pages of comprehensive bibliography are followed by a complete **author—**and **subject—index.** Besides, the location (orientation) of any expert treatise and the year of its appearance are easily made out at a glance.



In Part II, the Laws of the several States of the Union in North America, as relating to Occupational Diseases, the injuries they cause, and any pertinent material, are all represented. The plan and sub-divisions by which the situation can be ascertained for any one of the 48 states of the United States of America, are readily apprehended. In several cases corresponding paragraphs have cross-references. Especially valuable and complete is this legal survey for ascertaining any characteristic verdict (judgment) in respect of a pneumonoconiosis, showing the basis of the action, the conditions giving rise to the action, as well as the principle involved in the decision—for any state in the Union. The book closes with an alphabetical summary of the enterprises concerned in the juridical distinctions (or decisions.)

Examination of the book compels recognition of the extraordinary care and pains taken by the compilers. Random tests here and there show that for a space of 377 years this bibliography has attained a completeness not hitherto arrived at. Probably no branch of any other science has at its disposal so flawless and richly extensive a bibliography. It is authoritative not only for physicians and industrial experts, but also for physicists, engineers, and employers of industry. The format is entirely appropriate; for the arduous work involved the compilers should receive the fullest recognition.

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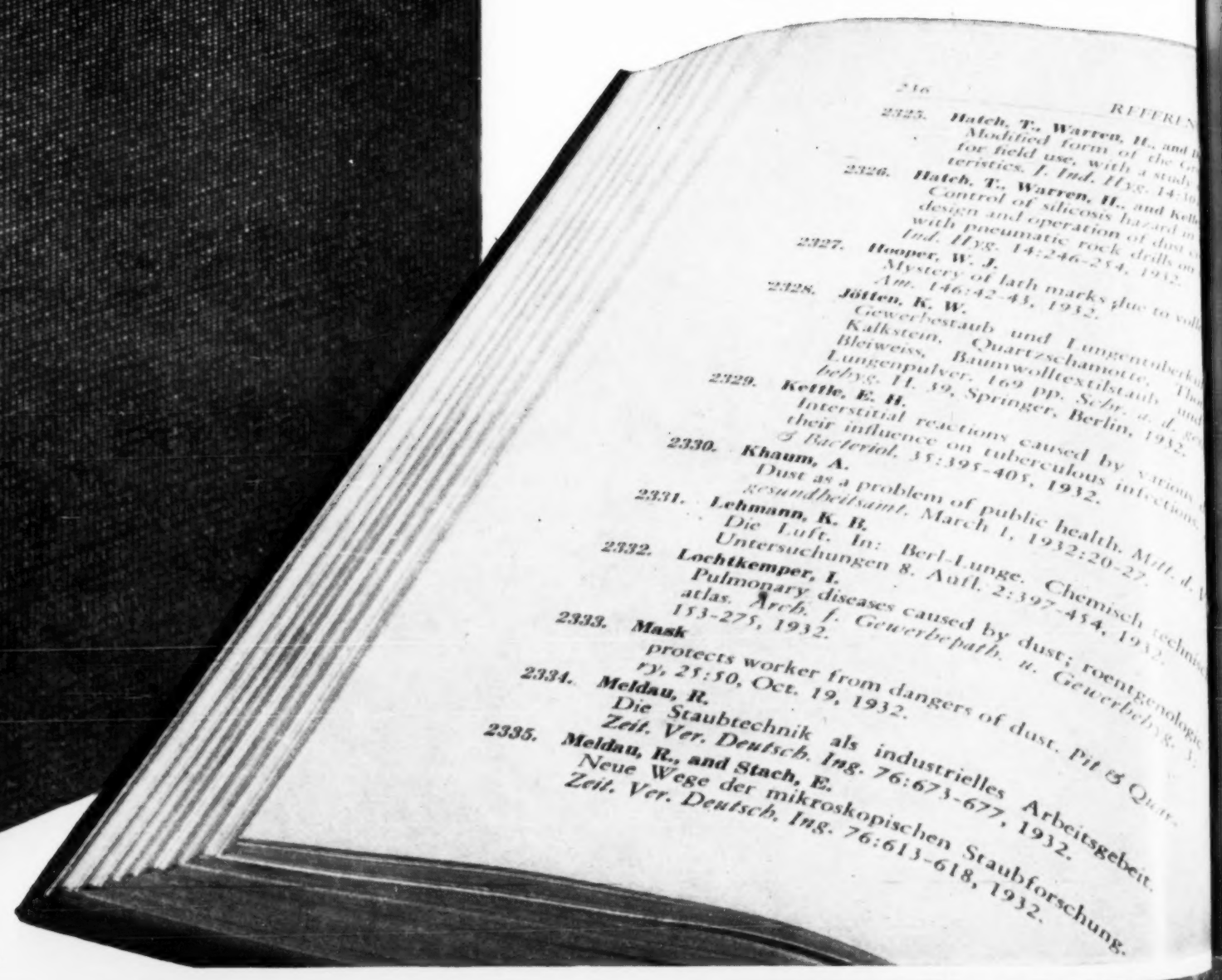
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This magazine is published to promote sound thought upon and concerning industrial medicine and traumatic surgery. To that end it will contain articles, news items, reports, digests and other presentations, together with editors' comments. The editorial policy is to encourage frank discussion. On this basis contributions are invited.

Industrial Medicine
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The editors will exercise care in checking on the accuracy of data printed, but in all other respects articles and opinions of which expression is allowed are the opinions of their authors—the editors reserving in all cases the right to comment on the same, in the current or any subsequent issues, as they may be inclined.

The Journal of Traumatic Surgery and Occupational Diseases

The Law, the Science and the Economics of Industrial Health

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The Trends of Industrial Medicine

—*With Particular Reference to the Prevention of Occupational Diseases*—

by

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I HAVE been privileged to make two surveys of medicine in industry, about 20 years apart, and I have followed this type of practice through the various phases of its development.* There have been four of these. We are now in the fifth. They can be named according to their outstanding features:

Surgical. Medical.
Job Placement.
Physical Supervision, and now
Engineering Control.

Each phase has added important functions, and the total result is an effective plan for health maintenance, a powerful preventive medical program of great social, economic and industrial importance.

The Surgical Phase

THIS was the genesis. It was characterized wholly by the care of injuries chiefly serious. Usually physicians were selected because of surgical experience, but often for availability. It was an emergency service. Minor injuries received little or no care until compensation laws made their neglect expensive, mostly because of infections. This suggested the need of early care, but to send all employees having minor injuries out for treatment encroached upon their productive time and introduced a new item of expense which was later offset by bringing doctors into the plants. They were given modest quarters and served for stated periods daily. Their presence in plants led to the assumption of other duties, to be discussed later, and they gradually became less obviously surgeons and more physicians with an aptitude for industrial practice.

Reontgenologists and orthopedic surgeons were ready to accept this opportunity, and their influence upon the treatment of industrial fractures has been invaluable. More recently, the physiotherapists have entered the field and now come the specialists in pulmonary diseases, the cardiologists and others. The eye surgeons, of course, have long been associated with industry, and so have the dentists in the care of injuries.

It is now regarded as good practice for plant physicians to have able consulting staffs. They know when to call in consultation and their patients receive better care. Industry is better served. In seeking consultation, however, it is important for plant physicians to bear in mind that specialists rarely have the industrial point of view. They do not fully realize, for instance, the value of the shortest possible disability periods and the necessity of fitting permanently disabled workmen into gainful occupations.

It is, therefore, evident that plant physicians are responsible for the correlation of all special services with their own to the end that the working men and industry may both be best served. This places plant physicians in a position of great opportunity as well as considerable responsibility, and no doubt the American College of Surgeons had this in mind when it became interested in the industrial field. But the query arises: How is it possible for plant doctors who refer all their surgical work to enjoy the maximum benefits of compliance with the College program if they themselves are not permitted to become fellows?

The *trend* is toward more consultations, better relations between consultants and plant doctors, better care of injuries, shorter disability periods and less permanent impairments.

The Medical Phase

THIS phase has been featured by the treatment of employees, often gratuitously, for sickness having no relation to industry. The development was natural. For example, an employee is taken suddenly sick. It is an emergency. The doctor is at hand and is called. He may continue treatment, thereby acquiring the patient, or he may call in the workman's own doctor after giving temporary aid. Another example: A workman has some trivial symptom. He probably would not see his own doctor but the plant physician is there—why not see him? The doctor treats him and tells him to see his own doctor later. He does not. He comes back. And the cycle continues.

* Presented at the Twenty-first Annual Meeting of the American Association of Industrial Physicians and Surgeons, Atlantic City, N. J., October 8, 1936.

From such small beginnings evolved an aspect of medical practice in industry which became exceedingly irritating to the medical profession and of no profit to plant doctors.

The influence of benefit societies and contract practice in mining and isolated industrial communities may be ignored because these types of practice are not, strictly speaking, industrial medicine and do not represent average conditions.

There is no point in discussing this phase further than to say that the trend toward non-industrial medical service is definitely stopped. Industry has neither intention nor desire to compete with private medical practice, but it does wish plant physicians to be medical counselors to workmen, assist them to obtain early consultations with their own physicians, and do everything within their power to sustain existing good relations between workmen and their family doctors.

In carrying out this policy of helpfulness, industrial physicians are rendering a service of much value to working men, are becoming important sources of satisfactory personnel relations for the employer, and are proving to be esteemed allies of the general medical profession.

The *trend* is toward cooperation with the medical profession. The effect is earlier and better medical care for the working people.

Job Placement

THIS phase is concerned with physical examinations and suitable placement of applicants for work. The theory is sound. With suitable placement workmen can be occupied more profitably and with less physical deterioration than if employed without consideration to fitness. But the medical profession was not prepared.

Plant physicians had been treating patients mostly for surgical conditions. As in private practice, they had been accustomed to wait for their patients to come into their plant offices or to send for them. Having few occasions to go into the plants, they knew little or nothing about working conditions and their relation to health. To them job placement was a theory capable of development possibly, but at the time beyond practical application.

Furthermore, it was difficult for doctors to adjust themselves to the circumstances under which examinations had to be made—nervous, impatient applicants in line and foremen calling for men. It was all too confusing for good medical work. The examinations became superficial. First aid men were substituted for doctors in the examining rooms. The pre-employment inspection had arrived, its principal effect seemingly the rejection of the obviously unfit.

Parenthetically it may be said that suitable placement of applicants and the benefits to them and industry from effective placement justify the rejection of a certain percentage as unfit, but it is hardly fair for a first aid man to be the judge when the finest of medical opinion is sometimes necessary.

This perversion of the theory of pre-employment physical examinations was a serious retardment. Nevertheless, being fundamentally sound it persisted. Now there is ample justification.

Plant doctors are studying working conditions, becoming familiar with occupational requirements in relation to health, making better physical examinations, and exercising the judgment of experience in rejection and placement.

Incidentally the use of x-rays and other diagnostic aids is symbolic of this changed status. The *trend* is toward better diagnosis, more effective placement and fewer rejections.

This phase of industrial medicine has a reaction upon physicians themselves which warrants emphasis. Pre-employment examinations are their introductions to new employees who may later become their patients and subject to decisions on the validity of claims. It behooves the doctors to win their confidence, and the examining room is the place to do so. New workmen are just as quick to recognize and respond to sincerity, medical skill and good judgment as any other group of people. The inference is clear. To be worthy of the confidence and respect of new employees, plant doctors must give them the same consideration they would in private practice. This is a fundamental principle in industrial medical practice.

Physical Supervision

THIS phase has logically followed job placement. The object is to keep workmen suitably occupied throughout employment and to give them the best possible opportunities for the maintenance of their health. It is featured by the various periodic examinations and consultations that plant physicians find essential to health maintenance.

The examinations are made on such occasions as doctors deem necessary, depending upon the physical condition of workmen at the time of employment and the nature of their work.

Employees are transferred to more suitable occupations when not well placed and referred to their own physicians for the treatment of non-industrial ailments.

Further details are not essential. The point is this. Physical supervision is becoming more and more a matter of accurate diagnosis and to that end diagnostic accessories are being used in constantly increasing numbers for the study of chest conditions, blood disorders, deficiencies of the cardio-vascular system, disturbances in the endocrine system, metabolism, allergy and various others. In short, the *trend* of physical supervision is toward periodic physical examinations, sufficiently complete to assure accuracy in diagnosis and often enough to assure early recognition of disease. This is without competition with the medical profession. It is complimental to general medical service among the working population.

Engineering Control

DURING the development of the preceding phases attention was centered upon the workmen, their treatment when injured, temporary care and advice when sick, selection for employment and subsequent physical supervision. The object was to assist them in the maintenance of their health.

The present phase is concerned with conditions under which they work and the materials they work with. Are these harmful to health? If so, how can they be made safe?

For the answer the doctors have moved out into the plants. They are seeking sources of disease, studying them and making suggestions for

their control. This is preventive medicine. It rounds out the program of industrial hygiene.

I have the impression that industry has been moving steadily in this direction for some time in advance of the medical profession. The industrial trend has been toward the removal of dusts, fumes, mists, vapors and gases which are the principal sources of disease, the substitution of non-toxic materials where practicable, and improvement in general factory conditions.

A great variety of chemicals, more or less toxic, are used in industry and new ones of undetermined toxicity are appearing from time to itme. The old method of installing new processes and using them without consideration to possible harmful effects is giving way to the more orderly procedure of first submitting them to the scrutiny of the toxicologist and the engineer.

When I say the doctors have moved out into the plants it is with the reluctant confession that they have no claims to priority. The fact is the toxicologists and the engineers have moved into the plants and are taking the doctors with them—the toxicologists to determine sources of disease, the engineers to determine nature and extent of exposures, the doctors to determine nature and extent of effects. From their joint efforts is to be expected an excellent industrial hygiene program with control of occupational diseases and reduction in sicknesses not fully occupational in character but possibly influenced by working environments: pneumonia, tuberculosis, degenerative diseases, etc.

It matters not whence comes the impetus for this trend. It may be the result of a desire for better working conditions on the part of employers and employees alike. It may be the result of an aroused public interest or the tendency of legislatures to compel compensation for occupational diseases. The fact is, the trend is here. It is toward the hygienic and engineering control of occupational diseases. It is featured by the affiliation of corporations with universities for special toxicological researches, the endowment of laboratories, and the creation of industrial hygiene engineering services in plants.

Medical Service in General Motors

MEDICAL service in General Motors Corporation is a function of personnel management. As such, it is expected to make substantial contributions to both the employee group and management by holding down lost time and building up goodwill without, however, infringing upon the rights of physicians in private practice.

Because of their specialized knowledge, plant physicians are left somewhat to their own devices in fitting themselves into plant organizations. They have to sell themselves to management and workmen alike. They must not over-sell to either. If they do, they raise the level of medical service above that justified as good business practice and at the same time are apt to wander into the field which properly belongs to private practice.

The doctors are expected to sell themselves to management by the use of good judgment, knowledge of plant conditions, the confidence workmen have in them and their professional skill. They may sell themselves to the workmen by their ability as physicians, their familiarity with conditions ont in the plants, their personal relations with the workmen and the respect they get from physicians in private practice, their medical societies, etc. In establishing themselves they have the same problems as physicians in private practice. They must treat their patients with the same consideration and professional thoroughness.

Doctors have made themselves valuable to industry. They came originally to treat injuries. They increased their usefulness by giving temporary care for trivial ailments and emergency sickness and by helping applicants to obtain employment suitable to their physical conditions.

Now that plant doctors are venturing further into the field of preventive medicine, the following program has been designed for their guidance. It is based upon the theory that to maintain health, conditions which may cause disease must be located and controlled, suitable workmen must be employed and they must receive appropriate medical supervision. At present emphasis is on the control of exposures but medical supervision is equally important.

It should be noted that the term "exposures" has been substituted for "hazards" as being more appropriate to the progress made in furnishing healthy working conditions.

1. LOCATION OF EXPOSURES:

This is a function of chemical engineering, sometimes called industrial hygiene engineering. The purpose is to locate all possible sources of disease, ascertain their nature and determine their extent. Some units have chemical engineering services of their own. For those who do not the Department of Industrial Relations of the Central Office is prepared to furnish it.

Surveys are made with the assistance of the plant physician and the findings are used by them as a basis for physical supervision.

2. CONTROL OF EXPOSURES:

Exposures are usually in the form of dusts, fumes, mists, vapors and gases. Control is obtained preferably by changes in process or substitutions where possible. If such are not possible, air contaminants are removed through exhaust ventilation, supplemented by respiratory protective devices if necessary. This is not a medical function, but when plant physicians have determined that conditions are such as may cause disease their recommendations are given appropriate consideration.

3. MAINTENANCE OF CONTROL:

The ordinary methods of inspecting safety equipment, exhaust ventilation, etc., are supplemented by:

(a) Monthly medical inspections of sanitary and working conditions with special attention to possible sources of disease.

(b) Special studies of dust and other air contaminants upon the recommendations of the doctors.

(c) Investigations of known cases of disease.

(d) Discussion with the physicians concerning the possibility of disease resulting from new processes or modifications in old. If there be such, appropriate measures are taken before installation or modification of operations.

4. PRE-EMPLOYMENT EXAMINATIONS:

It is desired that these be sufficiently complete for workmen to be placed in jobs they can do without impairment of health. The doctors' decisions are final.

5. Periodic Examinations:

New employees are examined frequently and if physical impairments attributable to or aggravated by occupation be found they are transferred. These examinations of new employees are made frequently enough and with sufficient thoroughness to assure recognition of disease in its early stages. All other employees are examined at suitable intervals depending upon the nature of their work or the presence of physical handicaps. The frequency of these examinations is fixed by the doctors, according to their judgment, sufficiently often to protect workmen against ill effects. All workmen not otherwise examined are examined annually.

6. Instruction:

Employees are given instructions by physicians where necessary for the protection of their health.

7: Records:

Records are essential to the maintenance of health and defense against unfair claims. They are, therefore, complete as to facts concerning each injury, illness and the findings of all physical examinations.

The foregoing is the outline of a health maintenance program only. It is supplemental to the usual functions of industrial medicine, the treatment of injuries, etc. The object is to secure a well rounded medical service in each unit without infringement upon the rights of physicians in private practice.

There are two principles in the program:

(a) The physicians shall make periodic plant inspections, and

(b) They shall make physical examinations of applicants for employment and periodically thereafter of all workmen.

The purpose of the plant inspections is to acquaint the physician with the conditions under which workmen are employed in order that:

(a) They may have specific knowledge of possible disease exposures, and

(b) They may aid the men to obtain work suitable to their physical conditions.

The purpose of physical examinations is to acquaint the doctors with the conditions of health, body and mind of the workmen in order that:

(a) They may assist them to maintain their health and productiveness, and

(b) They may have information for the making of fair decisions on claims.

It is evident that plant inspections and physical examinations must be made by the doctors themselves, otherwise the information has little medical value.

For defense against unfair claims it is not necessary for plant doctors to be partisan, nor is it necessary for them to build up defense. Adjudication of compensation is not their function. It is their duty, however, to have accurate, complete records as to the physical conditions of employees and such injuries and diseases as may occur in order that claims can be fairly judged.

General Motors has no centralized medical control, but a central consultation service is available to all units, covering the general principles of plant medical service, organization, quarters, equipment and functions.

Because of this decentralization, the doctors are their own medical authorities. They have been selected because of confidnce in them, and so long as that confidence is warranted their careers are assured.

The qualities that assure permanency are professional knowledge and skill in applying it to the needs of industry. The latter is measured largely by their capacity to make and retain the friendship of the workmen. The doctors are the closest point of contact between management and men. They meet them in the examination rooms and on numerous occasions thereafter when workmen are receptive. All are excellent opportunities for physicians to cement the good feeling that General Motors desires to exist between management and men.

Governmental Cooperation

THE United States Public Health Service has long been active in industrial hygiene. Its record of achievements is a credit to the Service and American industry. A few of the states also have been active in this field, and now there is a trend on the part of others and city governments as well toward the creation of Industrial Hygiene Departments usually in connection with their Health Departments.

Educational Relations

CERTAIN institutions of learning, realizing the insufficiency of information on occupational diseases, have been pioneering in reasearch and gradually building up knowledge in this direction. But now comes a demand for trained industrial hygienists, engineers and toxicologists, and in response to it a number of other universities are entering the field.

The difficulty is to find personnel, adequately informed and trained, and this applies also to the governmental agencies. Incidentally, industry itself has great difficulty in finding physicians qualified in industrial hygiene.

On the background of research, institutions of learning are now organizing training courses for physicians and others who desire to enter the field. This trend is a most wholesome evidence that industrial hygiene has arrived, and indicates more accurately than any other development the general trend toward well organized control of occupational diseases.

Relations to Medical Profession

AS INDUSTRIAL medicine has moved through these various phases there has come a constantly changing attitude on the part of the general profession towards physicians in industry.

At first the profession was indifferent, then irritated and later antagonistic, largely for the reason that industrial medicine seemed to be a serious competitor and was thought capable of disturbing the fine feeling that has existed between physicians and their patients from time immemorial.

It has appeared, however, that industrial physicians are in reality allies of the general medical profession and that the two have a common interest in the welfare of the public. Industrial physicians have their field, the general practitioners and specialists theirs, and there is no conflict. On the other hand, there are excellent opportunities for cooperation to the benefit of the general profession, the industrial physicians and the employed classes.

Relations to the Working Men

I CAN well remember the time when workmen looked down upon plant doctors. They had no confidence in their ability, or accepted them only with toleration.

When pre-employment examinations were introduced, labor received the idea distrustfully, because of misunderstanding, but as it became more evident that the purpose of physical examinations, both pre-employment and periodic, is to assist working men to conserve their health and maintain their productivity, the attitude toward plant doctors changed. They are regarded as friends and allies.

This wholesome change has been brought about by adherence to a principle which I believe industrial physicians, generally speaking, have followed since the beginning, namely that to serve the employer best they must serve the working man as they would their own private patients with courtesy, professional honesty and skill.

Relations to Industry

WITH these changes in industrial medicine has come a change in the attitude of industry toward plant physicians. Industry now recognizes their ability to be helpful, the important place they take in the lives of the working men and their capacity to create goodwill.

On the basis of this alone, plant medical service is an important function of personnel management. Furthermore, industry respects medicine and the allied branches of engineering and toxicology because of their ability to minimize disability periods, control occupational diseases, and through the maintenance of health facilitate production.

Medicine is firmly entrenched in industry and to physicians happily placed offers opportunities for satisfactory medical cereers with a degree of economic stability somewhat above the average for the profession.

Discussion:

DR. GEORGE H. GEHRMANN: The inception of industrial medicine was quite a number of years before the passage of compensation laws but their enactment gave impetus to its development. At first there were the contract surgeons, who were almost pariahs to their medical societies, but they nevertheless, supplied the service that was needed in industry and on the whole did a good job, so that they became indispensable to and a part of the industry. Of late years the contract surgeon has developed into the industrial surgeon and is now so recognized by the A.M.A. and the American College of Surgeons. To him can be given the credit for the instituting of physical examinations, pre-employment and periodic, and the keeping of accurate and complete accident and safety records. The advent of occupational disease laws enlarges the scope of the industrial surgeons activities, as he will now have to devote considerable time and thought to the prevention of diseases due to occupation by removing or mitigating the health hazards of the job. An accurate diagnosis must be based upon an accurate examination.

There is quite a difference between an examination for an industry and one made by a general practitioner. History in an examination for an industry is of no value. Keen perception of physical abnormalities and the use of laboratory aids, x-rays, electrocardiograms, etc., must be substituted for the history the patient gives us.

The ideal method of starting a new operation is to know its toxicology. Know what to look for; its symptoms and effects upon the organism. It is essential to know and have an accurate record of the exact physical condition of each employee. Physical examinations must be complete, thorough and accurate. It takes quite some time to make one. With us, one doctor examines 12 persons per day. The thought I want to leave with you is: you must know the health hazards and the toxicology of the operation.

DR. OTTO P. GEIER: Do we, the industrial suregons at large, find it as easy to do the things described by Dr. Selby as Dr. Selby does? No examination or re-examination can give us information of exact conditions in our employees as accurately as will the frequent visits of the employee to the medical department for injuries or minor illnesses. It is by these frequent contacts that we get to know them.

Industry is making these examinations for sound economic reasons, and it cannot afford to do it half-way. They must be thorough and accurate. Then there arises the question: When shall we stop? So as not to infringe upon the rights or trespass upon the domain of the private practitioner.

DR. A. M. HARVEY: Physical examinations must be emphasized. Periodic examinations are absolutely necessary. May it not be best to open up the medical department completely for the employees?

DR. L. D. BRISTOL: One thing I have in mind is how much the advance of industrial hygiene is indebted to the work done by our public agencies, especially the United States Public Health Service. There is still a great need for the inculcating of the principles of industrial hygiene among the smaller industries.

DR. SELBY, (closing): Much credit must be given to the older men in industrial medical service, for they have blazed the trails and opened the way for those who are to follow. But there is still much to be done to place industrial medicine upon a sound, stable basis.

Industrial medicine has no business or desire to encroach upon the domain of the private practitioner.

Traumatic Shock

By GEORGE H. MARCY, M.D.,
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TRAUMATIC shock was chosen as the subject of this paper for two reasons: *First*—because the great majority of deaths which we see in the capacity of railroad surgeons are the result of this condition; and *second*—because the authorities, in spite of a great deal of brilliant experimental investigation within the past few years, are still unable to agree on a theory which will adequately explain the underlying physiology; in fact, within the past few years the British honors[1] for outstanding work in medical science were divided between two men who held opposite opinions concerning traumatic shock.*

This paper will be limited to a discussion of secondary shock, using that term as first employed by James Latta[2] in 1795. In other words, we will not attempt to explain the immediate or primary shock, which is probably of neurogenic origin. Cowell[3] defines the syndrome as "the clinical condition which follows an injury producing depressed vitality, associated with lowered blood pressure, deficient circulating fluid, diminished intracellular oxygenation, and reduced body temperature". Neither this nor any other definition gives as clear a conception of the actual condition as a description of the "clinical picture", such as given us by John Collins Warren[4].

"A patient is brought into the hospital with a compound comminuted fracture, or with a dislocation of the hip joint added to other injuries, where the bleeding has been slight. As the litter is gently deposited on the floor, he makes no effort to move or look about him. He lies staring at the

* Presented at the Thirteenth Annual Meeting of the Association of Surgeons of the New York Central System, Detroit, Mich., October 13-15, 1936.

surgeon with an expression of complete indifference as to his condition. There is no movement of the muscles of the face; the eyes, which are deep sunken in their sockets, have a weird, uncanny look. The features are pinched and the face shrunken. A cold, clammy sweat exudes from the pores of the skin, which has an appearance of profound anemia. The lips are bloodless, and the fingers and nails are blue. The pulse is almost imperceptible; a weak thread-like stream may, however, be detected in the radial artery. The thermometer placed in the rectum registers 96 to 97° F. The muscles are not paralyzed anywhere, but the patient seems disinclined to make any muscular effort. Even respiratory movements seem for the time to be reduced to a minimum. Occasionally the patient may throw about one of his limbs and give vent to a hoarse, weak groan. There is no insensibility, but he is strangely apethetic and seems to realize but imperfectly the full meaning of the questions put to him. . . . The pulse grows feebler and finally disappears; and 'this momentary pause in the act of death' is soon followed by the grim reality. A postmortem examination reveals no visible changes in the internal organs."

This is a familiar picture and a typical example of shock following traumatism—a condition well known to surgeons to come on after some extremely painful and mangling injury. It is especially common following injuries which involve the exposure and mishandling of the abdominal organs, and practically the same complex appears after extensive burns.

In discussing the etiology of a condition so complex and so beset by hypotheses, it may well to start by briefly outlining the various theories which were accepted by outstanding men of their time. Hippocrates[5] did not express a theory of shock, but stated: "Wounds are very dangerous when there is some large nerve, vein, or artery injured, for the convulsion and the flow of escaping venal or arterial blood prostrates the wounded one and exhausts his strength". Most of the earlier explanations offered were vague and not worthy of discussion with our modern understanding of physiology. For example, John Hunter[6], who was well aware of the dangers of diminished blood volume, advocated bleeding. We will, therefore, proceed with the more modern theories.

Vasomotor Paralysis

AFTER the discovery of the sympathetic control of the blood vessels by Claude Bernard in 1852, Weir Mitchell, Moorhouse and Keen[7] were the first to regard shock as a reflex vasomotor paralysis. They believed that there was a loss of vasomotor tone, allowing a large quantity of blood to stagnate in the relaxed vessels, chiefly those of the splanchnic area. From the tapping experiments performed on frogs by Goltz, Fischer[8] reasoned that injury caused a reflex paralysis of the vasomotor nerves with a fall in blood pressure and dilatation of the great veins of the visceral regions, and in this way explained the circulatory failure occurring in connection with injuries both of the abdomen and other parts of the body.

The first evidence against this theory was the discovery of Loven in 1866, that stimulation of the central end of the cerebrospinal nerve caused elevation instead of lowering of the blood pressure. Moreover, surgeons who have had extensive experience in the recent great war and who have performed many abdominal operations on patients suffering from wound shock, testify that they have not found any evidence of splanchnic congestion. This could not be an oversight on their part, for 2000 cc. of blood (which is the amount lost in shock) would cause a marked distention of the splanchnic vessels if added to them.

That the vasomotor mechanism exerts itself to its utmost in the attempt to maintain an efficient head of pressure during shock is indicated by evidence presented by R. M. Moore[9], together with observations of many other investigators. The one reservation necessary is that exposure or manipulation of the abdominal viscera leads to local congestion, and is not due to paralysis of the vasoconstrictor center.

Theory of Exhaustion

CRILE[10] advocated the theory of exhaustion—that an excess of sensory impulses from the injured field might cause circulatory failure through the exhaustion of both the vasomotor centers and the brain cells. Thus he assumed at first that failure of the blood pressure was the primary and sole cause of the symptoms. His experiments were on dogs, which were put in a condition of shock by crushing the paws, or burning the skin, or removing part of the integument, or stimulating afferent nerves and by manipulation of the intestines. On microscopic examination of the brain tissue of these animals Crile discovered a series of changes which were directly proportioned to the duration and intensity of the trauma, and characterized by hyperchromatism, chromatolysis, eccentricity of the nucleus, rupture of the membranes, and finally disintegration. The evidence of these histologic studies unfailingly supported the assumption that the essential lesions in traumatic shock are in the central nervous system, liver, and possibly the suprarenals. These changes Crile attributed to an extreme demand on the cells due to an excess of impulses, leading to their exhaustion and consequently shock.

This theory of exhaustion was very adequately refuted by Cannon[11], who believed these morphological changes to be the result rather than the cause of low blood pressure. He pointed out that alterations exactly identical to those described by Crile have been produced by repeated hemorrhage, and have been attributed to prolonged anoxemia. Mann[12] has stated: "It is impossible to reduce the anaesthetized animal to a state of shock by any degree of sensory stimulation, provided all hemorrhage is prevented and its abdomen is not opened." Moreover, as we have already noted, there is considerable evidence that vasomotor paralysis is not the situation in this condition or the cause of the low blood pressure. There is in fact an increased vasomotor tone.

Acapnia

IN 1908 Yandell Henderson[13] presented the hypothesis of acapnia, which is defined as an insufficiency of carbon dioxide in the tissues and fluid of the body. According to his observations acapnia has a two-fold influence upon the circulation: (1) It diminishes the activity of the cardio-inhibitory center. The heart rate varies inversely as the carbon dioxide content of the arterial blood. (2) If a condition of arterial acapnia is prolonged,

the tonus of the venous system is abolished due, not to alteration of the vasomotor nervous system but to a peripheral action dependent upon the carbon dioxide content of the tissue.

Anyone familiar with the clinical picture of the onset of shock will realize that this hypothesis is based on a false assumption, namely: that there is excessive breathing due to the pain of the injury. Moreover, voluntary hyperpnea does not cause this condition. Experimentally, various investigators have demonstrated that the low carbon dioxide content of the blood can occur without shock and can be the result rather than the cause of low blood pressure.

Theory of Inhibition

DURING the same year that Henderson formulated his theory, Meltzer[14] noted that dissection of the skin over the abdomen caused a cessation of normal peristalsis. On this observation he based his theory of reflex inhibition, assuming the reduction of sensibility likewise implied inhibition. He considered apathy, insensibility, and muscular relaxation as the primary cause of the low blood pressure. To use his own words: "the various injuries which are capable of bringing on shock do so by favoring the development of the inhibitory side of all the functions of the body. The predominance of inhibition makes its appearance at first in those functions which are of less immediate importance to life, and are, therefore, less insured by safeguards protecting their equilibrium. With increased injury the inhibition also spreads to the more vital and therefore better protected functions of the nervous system. The early inhibition, in the development of shock, of the functions of lesser importance might even be looked upon as being in a degree conservative measures for the protection of rather more important functions of animal life. The restfulness of the body, the painlessness, and mental indifference are certainly most desirable conditions in the management of shock of the more vital functions."

More recent work has not tended to further this as an explanation of the circulatory failure. The observations of Porter, Mann, Janeway, Ewing, and others, have been confirmed by eight experiments of Parsons and Phemister[15]. These investigators exposed and stimulated the sciatic nerve and other nerves by frequently repeated crushings with forceps and by the application of a faradic current. In no instance was there a resultant fall of blood pressure, but usually a rise due to the Loven reflex. In order to be more certain that stimulation of the somatic nerves played no role in the production of low blood pressure, experiments were performed on 10 animals, the limbs of some of which were denervated, while others were left intact. Hammering the denerved limb resulted in low blood pressure similar to that produced in the animals whose limbs were not denervated. The decrease in pressure was dependent and proportional to the amount of injury. Due to these and similar experiments the nervous theory is at present considered to have little support.

Fat Embolism

ANOTHER theory which has caused much discussion and could not be left out of a paper of this kind, is that of fat embolism of the vasomotor center. W. T. Porter[16] published a paper to demonstrate that this was a previously unknown cause of early shock. His reasoning was based on the well-known facts that (1) shock is most frequent after shell wounds of the long bones; and (2) injuries of the long bones cause fat embolism. He argued against fat embolism of the lungs as being the important factor, because obstruction as great or greater observed in pneumonia or produced experimentally had no such effect.

In discussing the theory of fat embolism Cannon[11] first attacked it from the common sense point of view. He thought it very unlikely that the vasomotor center would be exclusively picked out in the floor of the fourth ventricle amid so many other vitally important centers. Other observers have pointed out the facts that (1) there is a striking difference between the symptoms of shock and those of fat embolism; and (2) clinical and experimental evidence does not demonstrate fat emboli in the medulla. Probably the most recent verification of this fact was done by the above-mentioned Parsons and Phemister[15]. With these observations in mind we are justified in concluding that the formation of emboli from fat of the long bones is not the cause of shock.

Chemical and Adrenal Factors

BOTH hyperactivity, and hypo-activity of the adrenal gland has been suggested as a causation factor in shock. The only support of such a theory is an occasional incident of shock being caused by a practically bloodless operation on a patient with Addison's disease. This would suggest that the adrenal is instrumental in the maintenance of vascular tone. The bulk of experimental work, however, has shown that the adrenals do not secrete excessively in severe injury, and their removal as demonstrated by Mann[12] does not cause an immediate drop in blood pressure.

More recently the similarity of adrenal cortex insufficiency and shock have been pointed out, suggesting a common causative factor. Within the past two years, however, Freeman[17] has discovered an important dissimilarity in the fact that blood sugar is low in the former and normal or elevated in the latter. The adrenal cortex evidently is not the cause of shock, but plays a part in regulating the blood volume.

Traumatic Toxemia

FOR many years the most widely accepted theory has been that of traumatic toxemia, which was propounded by Cannon[11] in 1923 as the result of clinical and experimental experience during the Great War. That toxic substances found in the tissues could produce a phenomenon similar to shock was first indicated by the classical experiments of Heidenhain[18] in 1891. Their action is somewhat different from that of histamine which was demonstrated by Dale and Laidlow[19] to have a marked effect through the vasodilator action on the capillaries and arterioles. The mechanism is evidently a poisoning of the endothelial walls of the capillaries allowing an increased permeability and causing a dilatation of numerous capillary channels which under ordinary conditions do not function. Hill[20], Krough[21], Hooker[22] and Rich[23] have demonstrated such a dilatation when the local tissue needs demand more oxygen and after the

injection of histamine. Such an increased permeability would lead to a loss of plasma from the capillary channels. This was shown to be the case by the investigations of Cannon, Fraser and Hooper[24], Taylor [25], and Robertson and Bock[26], who demonstrated a concentration of the corpuscles in the capillaries as compared with the veins, while the ratio of corpuscles to plasma in the veins remained approximately normal. Both clinical and experimental shock amply confirmed these observations of the blood changes. It was on these facts that Cannon based his hypothesis.

Although traumatic toxemia has been rather generally accepted as an adequate explanation of the lowered blood pressure, much evidence has been reported by Blalock and Bradburn[27] which does not substantiate the findings of Cannon. Crile[28] performed his so-called symbiotic experiments as evidence against traumatic toxemia as an explanation. In the latter a carotid artery of one dog was anastomosed to a carotid artery of another, and in internal jugular vein of one to the internal jugular of the other. Hammering the limb of one dog had no effect on the blood pressure of the other, which seemed to show that there was no toxic substance in the blood of the traumatized limb. Other investigators have obtained results similar to those of the symbiotic experiments. Blalock[29] transfused dogs with blood taken from dogs whose blood pressure had been reduced by trauma. No evidence of a depressor substance was elicited. Moreover, he found no acceleration of the fall of blood pressure upon the return of venous blood from a traumatized limb. In order to be certain that no toxic substance was returning to the circulation a tourniquet was put around the limb exclusive of the femoral artery and vein after a resection of the femur had been performed. A marked fall was elicited only with removal of the clip from the femoral artery after traumatization of the extremity. Smith[30] has carried on similar studies. Recently Roome and Wilson[31] have been able to demonstrate only slight temporary effects from extracts of traumatized tissue.

Moreover, by injecting phenolsulphonephthalein and strychnine into the burned or traumatized area, Underhill[32] and more recently Daniel, Upchurch and Blalock[33] proved that absorption into the blood stream is extremely slow from the injured part suggesting a one-direction permeability of the blood vessels in that area. The latter[34] has also demonstrated that histamine causes an initial decline in blood pressure, followed by a decrease in the output of the heart whereas uncomplicated hemorrhage and trauma cause exactly the reverse.

Of course, these experiments do not prove that there is not an amount of toxic substance in the blood obtained from traumatized tissue which is too small to produce symptoms or have any destructive effect on the brain cells. However, they are evidence that such a depressant substance is not the cause of the shock syndrome.

Hemorrhage

WITH these discrepancies in mind, recent investigators have again attempted to explain secondary shock on a hemorrhage basis. To determine the amount of fluid lost from the circulation into the injured area, Blalock[29] and Parsons and Phemister[15] repeated the experiments of Cannon and Bayliss[35], compared the weights of the traumatized and non-traumatized limbs. The difference indicated the loss of approximately one-half of the total blood volume into the injured extremity, and was sufficiently great to account for the decline in the blood pressure; in fact, it was more than could be lost by hemorrhage to the outside without causing death.

The fluid escaping into the tissues as the result of trauma to an extremity or intestines, or as the result of a burn, has been shown by Beard and Blalock[36] to be almost identical in composition to the plasma in the blood vessels. This is important, because the protein maintains to a large extent the osmotic pressure in the blood vessels. The natural supposition that loss of plasma produces an increased concentration of the red blood cells has been proven clinically and experimentally [29 30].

The belief is current that this concentration is one of the striking differences of shock and hemorrhage, as the latter is said to be associated with the dilution of the blood. Blalock[37] considered this an erroneous belief, because the blood pressure of those animals put in a condition of shock by hemorrhage did not remain low for an extended period, due to the death of the animals. Brooks[38] therefore reduced the blood pressure by gradual withdrawal of blood, and kept the animal alive for an extended period by transfusion. Death occurred despite the fact that more blood was transfused than removed. There was a marked increase in the concentration of red blood cells, and there was capillary congestion and hemorrhage in the tissues. These changes were found[39] to be proportional to the length of time of the low blood pressure. It therefore seems reasonable to assume that loss of circulating fluid causes the low blood pressure and insufficient oxygenation of the tissues, which in turn produced a change in all the tissues of the body, with increased loss of fluid and a negative response to transfusion.

Prevention and Treatment

REGARDLESS of the theory one accepts as an explanation of traumatic shock, the prevention and treatment remain the same. The most important feature of prevention is immediate first aid to the injured. This consists of splinting the injured part if fractured, of stopping hemorrhage by tourniquet or other means, of maintaining the body temperature by blankets, or insuring complete rest aided by morphine, and venoclysis. In the presence of a low or declining blood pressure, no manipulation or operative procedure should be done, or anesthetic administered. If it is imperative that something of this kind be done, local or gas oxygen anesthesia is preferrable to ether or chloroform or spinal, and transfusion should precede any such procedure.

Once the condition of shock has developed, the aim of treatment is to cause an increase in the volume of the circulating blood. Vasoconstrictor drugs, such as adrenalin, are only temporarily beneficial, although Heuer and Andrus[40] have reported some advantage in using adrenal cortical extract with transfusion. The ideal treatment would be to introduce into the blood stream some fluid that would remain there, thus increasing both the blood volume and pressure. Unfortunately the intravenous injection of the various fluids that are commonly used in the treatment of shock is associated with not only the loss of the greater part of

the fluid injected, but also with a decrease in the protein content of the plasma remaining in the vessels. Beard, Weinstein and Blalock[41] have demonstrated that this decrease in plasma protein did not occur only when whole blood or blood serum was introduced. It is for this reason that transfusion is considered the best treatment together with the supportive measures mentioned in the discussion of prevention. Since obtaining a suitable donor takes time and there is a concentration of the red blood cells, the immediate introduction of solutions of saline, glucose, acacia or the like, is indicated to shorten the duration of the low blood pressure, and dilute the concentrated red blood cells.

Conclusions

IN CONCLUSION, I must admit that one is in error if he tries to explain by one theory alone all the instances of shock. However, the experimental evidence at present points to the confirmation of the dictum uttered by the older generation of surgeons that "shock is hemorrhage and hemorrhage is shock". The best treatment we have for the condition is transfusion, but even this is of no avail for shock which has persisted for several hours.

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Medical Services in Industry

THE list of medical services in industry in the United States and Canada, approved by the American College of Surgeons as of October 1, 1936, is contained in the "Approval Number" of the Bulletin of the American College of Surgeons.* Approval is granted on the basis of compliance with the Minimum Standard for Medical Service in Industry formulated by the American College of Surgeons. The list also indicates provisional approval, which signifies that the medical services so designated have accepted the minimum requirements of the College standard and are endeavoring to put them into effect, but for lack of time or other acceptable reasons, have not been able to do so in every detail. A Certificate of Approval is granted to an industrial establishment in which the medical organization and service is fully approved and is of such a nature as to give reasonable assurance of continued compliance with the Minimum Standard.

The American College of Surgeons early recognized the need for better organization and service for the care of the ill and injured in industry and for the elimination or control of the health hazards that exist therein. Consequently a Committee on Industrial Medicine and Traumatic Surgery, which consisted of representatives of the College, of medical departments of insurance carriers, and of other industrial organizations, was appointed for the purpose of studying and improving this situation. Surveys of medical departments and services in industry were first made at selected points in the United States to secure unbiased, first-hand information of actual conditions and to secure a basis on which could be formulated definite conclusions and recommendations for the correction of existing deficiencies in the care of the health and injuries of industrial workers.

These early surveys revealed that industrial injuries generally had not been cared for by those better qualified in surgery; that many industrial preventive health measures had not been instituted by employers; that many of the physicians serving industry served on a curative basis only, and that they were little interested in the administrative and preventive phases of industrial medicine; that most of the smaller industrial establishments lacked adequate medical service; and that much constructive work could be done in this field by correcting the conditions responsible for this state of affairs which militated against the physical well-being of the industrial worker.

Based upon experience and the findings of these direct investigations the Committee on Industrial Medicine and Traumatic Surgery of the College formulated a Minimum Standard for Medical Service in Industry which is applicable both to

* From the "Approval Number" of the Bulletin of the American College of Surgeons, October, 1936.

large and small establishments. This standard, which is included with this report, embodies the essential features of an adequate industrial medical and surgical service, and compliance therewith will serve the best interests of both employer and employee.

The practical application of this standard was begun by the College in 1931 by making personal surveys of industrial establishments on a nation-wide basis. The first list of approved medical services was published in 1933. To date 1,593 industrial establishments, representing approximately 5,000,000 employees, have been surveyed of which number 817, or approximately 51%, have been fully or provisionally approved. The surveys have been practically limited to organizations having 500 or more employees but it is contemplated to include smaller establishments in future surveys.

The industrial medical services are resurveyed at intervals and are considered for rating annually. An establishment once approved will not necessarily remain so. All establishments automatically come off of the approved list on October first and are immediately reconsidered for a new rating, each establishment being evaluated on its own merits.

At the Conference on Industrial Medicine and Traumatic Surgery of the Clinical Congress of the American College of Surgeons held in October of each year, public announcement is made of the approved medical services in industry, and the list is published. It is realized that there are other medical services worthy of recognition of which time has not yet permitted a survey. Many requests for information and surveys are received by the College to which there is compliance without expense to industry.

The spirit of this movement to make adequate medical service readily available in industry through effective organization transcends the honor of having an approved medical service. It provides an added incentive for growth and constructive action in the solution and handling of a problem that is both economic and humanitarian in nature. Certain stimuli such as recent occupational disease laws and the newer interpretations of social justice with related legislation have, of course, added impetus to the movement. In comparison to the safety movement in this country, however, industrial medicine has heretofore had a slumberous career. It is true that individuals and small groups of industrial physicians have done splendid pioneer work in this field, but until recently employers as a whole failed to sense the tempo of these efforts.

In the last few years distinct progress has been made in industrial medicine. By throwing the spotlight on the defects which exist in the various medical services, corrective measures eventually follow. Health hazards in the industrial plants are being eliminated or controlled. An increasing number of employers and insurance carriers are realizing that the best medical and surgical service is the cheapest. Furthermore, by giving industrial medicine the recognition that it deserves, the present industrial physicians and surgeons are stimulated towards greater effort and efficiency and other competent physicians and surgeons are being attracted to this type of work. In short, the care of the human machine in industry is being transferred from the obsolete repair department to the modern maintenance department.

Working Principles

IN ORDER that the Minimum Standard may be more readily understood, a brief explanation of its working principles is herewith presented according to its five clauses.

"1. The industrial establishment shall have an organized medical department or service with competent medical staff including consultants and also shall have adequate emergency, dispensary and hospital facilities and personnel to assure efficient care of the ill and injured."

Clause one deals with organization and facilities for the medical service. The workmen's compensation laws require that the employer shall provide the necessary medical and hospital care for injured workers. Obviously the plan for providing this care should be so well organized by the employer that the mechanism or procedure in caring for industrial injuries becomes automatic in nature and that it will insure efficient care by competent first aid attendants and surgeons. In order to be of greatest value, however, the scope of all industrial medical services should include both preventive and curative phases as follows:

1. Pre-employment physical examinations and the indicated periodic health examinations.
2. Efficient care of all industrial injuries and occupational diseases.
3. First aid and advice for employees suffering from non-industrial injuries and illnesses while on duty.
4. Medical supervision of plant sanitation and of all industrial health measures for employees.

These factors are all taken into consideration in granting approval to an industrial medical service.

The unsupervised use of first aid kits or supplies throughout an industrial plant should not be tolerated. Fewer and centralized first aid stations or dispensaries are preferable in order to insure adequate treatment and records.

The first aid attendants should be properly instructed by the physician in charge and should be under his supervision. Surgical procedures engaged in by many lay first aid attendants throughout the country are illegal and render the employer liable. These practices which also endanger the injured employee should be discontinued.

"2. Membership on the medical staff shall be restricted to physicians and surgeons who are (a) graduates from an acceptable medical school, with the degree of Doctor of Medicine, in good standing and licensed to practice in their respective states or provinces, (b) competent in the field of industrial medicine and traumatic surgery, (c) worthy in character and in matters of professional ethics; in the latter connection the practice of the division of fees, under any guise whatsoever, shall be prohibited."

This clause of the standard is self explanatory. The physical well-being of the employee is of prime consideration and all matters pertaining to the care of ill or injured workers or the related preventive health measures should be entrusted to a well qualified and licensed graduate of medicine.

"3. There shall be a system of accurate and complete records filed in an accessible manner,

such records to include particularly a report of injury or illness, description of physical findings, treatment, estimated period of disability, end results, as well as other information pertinent to the case or required by statute for Workmen's Compensation claims or other purposes."

Clause three, which deals with the necessity of maintaining adequate medical records, particularly of injuries, is readily appreciated by most industrialists. Further improvement can be made in this phase of industrial medical service. Our surveys have revealed, for instance, that one-third of all pre-employment physical examinations made in this country are not recorded or are incompletely recorded. Too often non-industrial injuries and illnesses of employees that are treated in the plant are not recorded. Furthermore, periodic analyses of the injury and illness experience and its costs are not made by all industrial organizations. This is like playing a game of baseball without totalling the score.

Statistical summaries and comparative studies of the injury and illness experience and its costs should be made at intervals. These studies will not only aid in evaluating the efficiency of the medical and safety services but they will also serve to demonstrate future objectives of such services by uncovering any weak points that may exist.

Physical examination records of employees should be treated as confidential information and should be filed under medical supervision and responsibility. This point is of utmost importance in order to provide records which are readily accessible to the physician for re-examinations and follow-up purposes, and in order to maintain the cooperation of the employees and their confidence in the medical service.

"4. Patients requiring hospitalization shall be sent to institutions approved by the American College of Surgeons."

It is of utmost importance to the employer and to the injured employee that they be assured of adequate care when hospitalization is needed. Since 1918 the American College of Surgeons has been engaged in the standardization of hospitals in the United States and Canada. A Minimum Standard for Hospitals was formulated which was adaptable to all types and sizes of institutions caring for the sick and injured. Hospitals have made splendid progress in carrying out these requirements not only because the public has demanded better service but because the Minimum Standard has aided in establishing a better environment for the physician and his co-workers in rendering better care to the patient. To date there are 2,577 approved hospitals in the United States and Canada.

"5. The medical department or service shall have general supervision over the sanitation of the plant and the health of all employees."

Clause five pertains to the responsibility for the medical supervision and service. Obviously medical matters in industry should be in competent medical hands. State supreme courts have ruled to the effect that corporations cannot practice medicine. This service should only be rendered by those properly qualified and licensed according to the law. The workmen's compensation law of New York specifies that all first aid stations or clinics participating in the treatment of industrial injuries must provide for medical supervision.

Industrial physicians should formulate and sign standing orders for the first aid or medical service in the plants which they serve. These regulations should be posted in the plant's first aid room or dispensary for the guidance of the first aid attendants. Periodic visits to the plant should be made by the physician to insure compliance with these regulations. Periodic inspection trips through the plant should also be made in cooperation with the safety and engineering departments in order to control or eliminate all health hazards. Written reports of such inspections including the indicated recommendations should be made to the proper plant official.

It is important that the industrial leaders confer the proper authority upon those to whom they entrust the responsibility for their medical service. In return they should demand results from their medical directors. By so doing a stimulus will be supplied that will lead to greater progress and to a more efficient medical service than industry has ever enjoyed heretofore.

Colles' Fracture

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THE wrist-joint is formed by the radius and triangular cartilage above and the scaphoid, semilunar, and cuneiform bones below.* These are joined by the anterior, posterior, internal and external lateral, and capsular ligaments.

The hand can be flexed, extended, abducted, adducted, supinated, and pronated.

The lower end of the radius is large and spongy, covered with a thin layer of compact tissue. Its anterior surface is concave, its posterior convex.

On its inner side is a concave articular facet for the reception of the head of the ulna. The interarticular triangular fibrocartilage serves as the main bond of union between the lower ends of the ulna and radius.

The radio-carpal articular surface slopes downward and outward to end in the styloid process. The articular surface is divided into two facets: the outer is triangular in shape, and articulates with scaphoid; the inner or larger is quadrilateral and articulates with the semilunar.

The ulna contributes but little to the support of the hand and does not enter directly into the wrist joint.

Fracture of the lower end of the radius is called Colles' fracture because it was correctly described by the Irish surgeon in 1814.

This fracture is usually incurred by indirect violence, such as the individual falling on the outstretched hand.

The distal fragment of the radius is usually displaced posteriorly, giving rise to the so-called "silver-fork deformity". The anterior radial curve is obliterated. Normally, the styloid process of the radius is on a plane lower than that of the styloid process of the ulna. In Colles' fracture, often the reverse is found. There is marked pain on pressure over the distal end of the radius. Often the fracture is impacted, and, therefore, no crepitus or false point of motion elicited. Supina-

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tion and pronation are either lost or impaired; otherwise, there is nothing characteristic about the limitation of the movements of the hand. Backward tilting, lateral displacement, rotation of the lower fragment, or a combination deformity may be found. Many of the fractures of the lower end of the radius are comminuted, with fracture lines extending into the wrist-joint.

If the individual falls on the dorsum of the flexed hand, the the distal fragment of the radius is displaced anteriorly, which causes so-called Smith fracture or a reversed Colles'. This fracture is difficult to retain.

If a few hours elapse prior to examination, swelling complicates the diagnosis.

It is not unusual to find a Colles' fracture without displacement, but localized tenderness is invariably present.

X-ray examination, both antero-posterior and lateral views, should be made prior to reduction—including doubtfully fractured wrists. It is important that the surgeon make a careful study of the films prior to instituting treatment. A second x-ray examination should be made after the fracture is reduced and a permanent dressing applied.

Personal experience favors early reduction under general anesthesia. Complete muscular relaxation is imperative. Roentgenogram indicates the manipulative procedure. If the fracture is impacted, the impaction must be broken up by traction plus to and fro movements. If this is not done, a painful wrist with a shortening of the radius together with a shifting of the carpus toward the radial side results. The fracture is usually reduced by increasing the deformity to unlock the fragments, by traction in ulnar deviation, by thumb pressure over the posterior aspect of the distal fragment, by digital pressure over the anterior aspect of the proximal fragment, and by flexing the hand. Summarized—increase the deformity and reverse the force. The properly reduced simple transverse fracture is easily retained. The hand can be placed in a neutral position and suitable splints or plaster of Paris applied. These splints can be safely removed about 10 days later and physiotherapy instituted. The splints are re-applied and the wrist immobilized so long as there is danger of lower fragment displacement.

The oblique fracture, easily reduced, but retained with difficulty. Flexion and ulnar deviation of the hand is the position of choice. The retentive apparatus must guard against supination and pronation because such movements may cause recurrence of displacement. Fracture reduction and retention are equally important. The normal anterior radial curve must be restored. The retentive apparatus should not immobilize or restrict the motion of the metacarpophalangeal joint. Free motion in the fingers and the thumb during the immobilization period shortens disability time and lessens likelihood of permanent partial impairment. Early active and passive motion of the wrist are required in the treatment of this fracture.

Reversed Colles' fracture is best treated by fixing the wrist with the hand in extension or dorsal flexion.

In a simple transverse fracture, the upward pull of the flexor and extensor muscles of the forearm tends to hold the fragments in position. However, if either fragment is comminuted on its posterior aspect, bony resistance to the tendon pull at this point is diminished, causing a backward tilting of the lower fragment. To avoid this, the wrist should be immobilized with the hand in flexion. This kind of fracture requires longer immobilization. Senility does not contraindicate the routine treatment.

The period of disability depends on the severity of the injury, the occupation, and the treatment. Many individuals with Colles' fracture return to the office the following day, while a laboring man, requiring full use of the injured hand, would probably be disabled seven or eight weeks.

Incomplete reduction or recurrence of backward tilting of the lower fragment will cause a characteristic deformity. The flexor tendons and the median nerve are firmly held in place by the annular ligament. As the fingers are moved, the flexor tendons pass over the sharp edges of the proximal fragment, which causes a tendosynovitis, a painful wrist, and limitation of motion in the fingers.

Tendosynovitis and arthritis often result from too long immobilization. Ischemic paralysis, also called Volkmann's contracture, may result from undue constriction of the forearm during the period of immobilization.

The forearm, wrist and hand should be examined for associated injuries, such as lacerations, causing compound fracture with all its hazards; fracture of the styloid process of the ulna or division of its ligament with a resulting prominence of the lower end of the ulna; injuries to the distal radio-ulnar articulation and interosseous membrane, which is accompanied by widening of the wrist; fracture of the scaphoid; dislocation of the semilunar; injury to the median nerve, tendons, and blood vessels. Supination and pronation impairment may result from distal radio-ulnar articulation damage.

Fracture of the shaft of both ulna and radius immediately above the wrist often has to be treated by open operation and internal fixation.

A compound fracture must receive primary attention. The wound should be cleansed thoroughly from the center outward so as to avoid additional contamination. Surrounding hair should be removed with a razor, the skin cleansed with soap and water, followed by ether and tincture of iodine or an equally effective antiseptic, all foreign bodies removed, and devitalized tissue fragments excised. If there is a protruding contaminated fragment of bone, it should be scrubbed and sterilized as far as possible before it is reduced. Often the damage to the soft parts is greater than the opening in the skin indicates. If in doubt, it is safer to extend the skin opening, cleanse the wound, remove the foreign bodies, control the hemorrhage, and do whatever else is required. Surgeons know how unfortunate it is to have a compound fracture infected; therefore, every effort should be made to prevent such complication. Dakin's solution is much used to sterilize. If the soft part damage is great, if the contamination has been extensive, or if there is appreciable capillary hemorrhage, primary closure should not be attempted. Mechanical cleansing of the wound is absolutely necessary.

In conclusion, best results are attained by proper reduction and fixation, together with early motion, both active and passive.

Silicosis

—A Review of the Literature—

by

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THE annals of medical and legal writings are fairly bursting their bindings in holding the literature that has been written on silicosis in the past few years. The precipitation of this almost sudden burst of interest is well known to those interested in the various phases of this problem. The number of people exposed to this single industrial hazard comprise one of the largest groups of our working people; the importance of the study of silicosis, therefore, cannot be judged too highly.

The term "pneumoconiosis" was first used by Zenker in the middle part of the nineteenth century and is derived from the Greek (pneumon—lung; konis—dust). Its literal definition embraces all lung diseases produced by the inhalation of all forms of dust which produce irritation to the lungs. This disease is called "silicosis" when it is due to breathing dust containing free silica; "anthracosis" when it is due to breathing coal dust; "siderosis" when it is due to breathing iron dust, either from ferric oxide dust or inhalation of ferrous oxide; "chalicosis" when it is due to breathing air rich in calcium salts; "asbestosis" when it is due to the inhalation of fine particles of asbestos which contain magnesium, calcium and silicate. The two most important and clinically recognized diseases produced by dust inhalation are silicosis and asbestosis the other forms of pneumonokoniosis lack complete definitions and correlation with pathological findings and chemical studies.

Some of the dusts responsible for the disease termed pneumonokoniosis are bituminous and anthracite coal, gold, silver, zinc, lead, copper, iron, sand, granite, sandstone, shale, silica flour and sand, feldspar, lava, silicon carbide, silundum, fibrox, French chalk, tremolite talc dust, pumice, mica, talc, asbestos, willemite, meerschaum, sodium silicate (water glass used as a filler in soap), hydraulic lime, soap stone, serpentine, building stone, aillmanite, topaz, fuller's earth, caolin, clays, ultra marine, agalite, rock dust in coal mines, cotton and textile dusts, garnet (used as watch bearings, in gem cutting, polishing and grinding), permite (used in water softening), slate, glass and many others.

Silicosis has been defined as a form of pneumoconiosis due to breathing air containing silica dust; it is characterized anatomically by generalized fibrotic changes and the development of miliary nodulation in both lungs and clinically by shortness of breath, decreased lung expansion, lessened capacity for work, increased susceptibility to tuberculosis, and characteristic x-ray findings.

HISTORICALLY, silicosis has been known by many for centuries, as is indicated by the literature. Hippocrates spoke of the metal digger as a man who breathed with difficulty and had other symptoms similar to those found in silicosis. Agricola, in 1557, stated that "some mines are very dry and the constant dust enters the blood and lungs, producing difficulty of breathing the Greeks call asthma. When the dust is corrosive, it ulcerates the lungs and produces consumption, hence it is that in the Carpathian mountains there are women who have married seven husbands, all of whom this dreadful disease has brought to an early grave." Lohneiss, in 1690, referring to miners, describes the effects on them as follows: "The dust and stone fall upon the lungs, the men have lung disease, breathe with difficulty, and at last take consumption." Bernardino Ramazzini, in 1700, published a book, De Morbis Artificum, which was the first of its kind in the history of occupational diseases and made special reference to stone-cutters' disease. In 1713 a British patent was granted for grinding flint by wet methods. Previously the flints were pounded dry, which "proved very destructive to mankind, so much that any person, ever so healthful and strong, working in that business cannot possibly survive over two years, occasioned by the dust sucked into his body by the air he breathes." In 1862 Dr. Peacock gave a report based on an examination of over 600 miners, in which he established the existence of miners' disease, distinguishing it from true phthisis, stating that "The quickness of pulse, the rapid and extreme emaciation, and the night perspiration so characteristic of true phthisis are generally absent or only slightly marked." In 1902 a committee, of which Dr. J. S. Haldane was a member, reinvestigated the causation of the high mortality of phthisis among Cornish tin miners, and decided that "so far as the Cornish miners are concerned it seems evident enough that stone dust, which they inhale, produces permanent injury to the lungs, gradually, in the case of ordinary miners, and rapidly in the case of machine-drill men. That the primary injury to the lung is due solely to the inhalation of dust would seem to be practically certain."

Dr. J. H. L. Cumpston reported in 1910 that the death rate from respiratory disease among all male non-miners over 15 years of age was 167 per 100,000, whereas the rate among miners was 539 per 100,000. The death rate due to tuberculosis among males over 15 years of age was six, whereas among miners it was 192 per 100,000. In New South Wales in 1921, among 6,538 miners 373 were found to have some incapacity due to the effects of dust on the lungs. In 1922 in New Zealand, 2,012 men were employed in gold mines, 1,438 of whom worked in quartz gold mines. During that same period 506 former gold miners received annual compensations for incapacity due to effects of silica dust.

More work has been done in South Africa on the study of silicosis than in any other country. This is due to the fact that a far greater number of men, from 12,000 to 15,000 whites and 180,000

blacks, in a limited region are exposed to rock dust containing a high percentage of silica.

For the years 1908 and 1909 the Miners' Phthisis Commission of South Africa considered that out of a mining population of 12,000 approximately 1,000 men in any one year would reach the stage of definite incapaciation. On account of the heavy morbidity from miners' phthisis, a Miners' Phthisis Medical Bureau was established in Johannesburg in 1916 under Government auspices. By periodic examination of miners actually employed, 738 were found to be suffering from simple silicosis in 1916-17, and 700 in 1917-18, 704 in 1918-19, 1,572 in 1919-20 (this year the anteprimary stage was included), 448 in 1920-21, and 434 in 1921-22.

In the Joplin district, in Missouri, 433 miners out of 720 examined had silicosis. In Butte, Montana, 432 out of 1,018 examined were also affected.

AMONG the occupations which contribute sufferers from silcosis may be mentioned mining, cement making, sand-blasting, stone cutting, blast furnace work, soap making and particularly the manufacture of abrasive soaps and powders, paper making where agalite is used in coating paper, the working of fine clay and other clays, the spray coating of plumbing fixtures, rock drilling, stone and metal polishing and grinding, sculpture, work in cotton and textile trades, the making of monuments and tombstones, manufacture of sandpaper, gem cutting and polishing, the glass industry, the pottery and porcelain trade, and the construction of railways, highways and tunnels.

Climate, season, soil, heredity, race, age or sex seem to have little or no effect upon the development of silicosis. It has been found that the incidence is higher among the young miners in districts where the percentage of free silica is high, and among older miners where the percentage of silica is low, age in itself probably is no great factor.

Individual disposition to silicosis varies to a great extent. Grave pathological changes may be found after a relatively short exposure to the dust and a relatively long latent period. Persons with a four-plus Wassermann reaction develop silicosis more quickly than the Wassermann negative group, and the disease runs a more rapid course in a syphilitic individual.

The finer the dust the more will be inhaled. Silica dust found in lungs varies from one-half micron up to 10 microns, but does not exceed 10 to 12 microns. In a South African mine where spraying the air had been resorted to in an effort to lessen the dust hazard, one droplet is reported as carrying over 800 particles, each smaller than two microns. Greater carrying power was noted from grindstones in Great Britain where droplets of two millimeters in diameter conveyed 4,383 particles each, with twenty-four microns as a maximum dimension. The minimum dosage is not known, but the condition is progressive and the symptoms of the disease may appear years after the worker has discontinued the exposure.

AS TO the methods of collecting dust, McNally says: "Various methods have been used in collecting samples. We formerly used the sugar-tube method, supplanting it by the Palmer water spray apparatus. The Bureau of Mines, at the Pittsburgh Station, made comparative tests of the various methods in use and a new instrument, the Impinger, was evolved. In this instrument, the air to be sampled is drawn through a glass tube and impinged at a high velocity into a graduated tube containing water. The dust is momentarily arrested, wetted by the collecting fluid, and in this manner trapped. The apparatus possesses an efficiency of 94 to 97.5% when sampling finely divided silica dust suspension at the rate of one cubic foot per minute." A portion of the collecting fluid is removed to a Sedgwick Rafter Counting Cell for microscopic count to determine the number of particles present. An eyepiece micrometer known as "Whipple Disk" can be placed in the eyepiece of any microscope in making the counts. The great objection to this form of apparatus is the weight, as one person cannot carry both parts of the apparatus and tripod.

The dust count should not exceed 10,000,000 particles per cubic foot when collected by the Greenburg Impinger Method, and counted with about 110 diameters magnification. This is equivalent to 300 particles per cubic centimeter (referring to silica dust exposure). This is a standard used in South Africa, Ontario, Wisconsin, and Oklahoma.

THE diagnosis of silicosis is established by a history of exposure to the inhalation of silica dust in occupation, by roentgenological examination and by physical examination.

In the embryological development of the lungs, the earliest indications in embryos are at 2.5 millimeters with 23 segments, with the formation of the laryngo-tracheal groove which runs lengthwise in the floor of the gut. The entodermal groove or ridge becomes in order the larynx, trachea, while the more rounded caudal end is the unpaired primondium of the lungs. The respiratory system is lined with epithelium, in this case entoderm which is strengthened and supported by other layers differentiated from the surrounding mesenchyme. The lungs expand into the coelon (pleural cavities) and in so doing gain a covering of visceral pleura surfaced with mesothelium. At the four-millimeter stage the lung bud begins to bifurcate and the respiratory organs are then represented by the laryngeal groove, the tubular trachea, and two lung buds or primary bronchi. Soon after the lung buds are formed, the right becomes larger and directed straighter caudal. At the seven-millimeter stage these stem bronchi give rise to two lateral bronchial buds on the right side and one on the left, thus the future pulmonary lobes are indicated. The bronchial buds continue to branch so that the tubular system in each lobe becomes more tree-like with the dorsal, ventral, lateral and median rami.

The lungs are formed by the ramifications of the bronchi and bronchioles, and their terminal expansions. The bronchioles with their terminal expansions form the primary units or lobules. In the primary unit the terminal bronchiole is continued on as the vestibule. The vestibule divides into from three to six atria, and each atrium divides again into two or more infundibula. In addition to those which open into the infundibula, there are a few alveoli in the walls of the atria and vestibules opening directly into these passages. The bronchi are lined throughout by ciliated columnar epithelium. There is a gradual

transition from ciliated columnar to cubical and non-ciliated epithelium in the terminal bronchioles. The cubical type of epithelium may be continued along the greater part of the vestibule. In the semi-expanded lung two types of lining cell may be identified in the alveoli. One appears as a flattened square, the other is cuboidal in shape. The cuboidal cells are known as "septal cells." They occur in groups very often covering the minute spaces between the capillaries. The alveolar phaagocytes are said to owe their origin in great part to the septal cells. An important structure in the walls of the bronchi is the circular muscular coat composed of plain muscle. It is continued along the bronchioles and sometimes along the whole length of the vestibules. The mesenchyme surrounding the bronchial tree ultimately becomes muscle, connective tissue and cartilage plates of the pulmonary walls. Into it grow blood vessels and nerve fibers. When the pleural cavities are completed, the mesothelial and connective tissue covering of the lungs becomes the visceral pleura. The corresponding layers lining the thoracic wall form the parietal pleura.

The lungs receive most of their blood from the pulmonary arteries whose branches accompany the bronchi and their branches as far as the respiratory bronchioles. Here they divide and a branch passes to each alveolar duct and is distributed in a capillary network over all the alveoli, which communicate with this duct. The capillaries of the pleura, the capillaries of the alveolar septa, and portions of the alveolar ducts go together to form the venules which are found in the interstitial connective tissue and fuse to form the pulmonary veins. In passing through the lung, the pulmonary artery is usually above and behind its acompanying bronchial tube while the vein is below and in front of it. The bronchial arteries and veins arise from the intercostal arteries and follow the bronchi and are distributed to the walls of the bronchi, their glands, and the interlobular connective tissue beneath the pleura. Most of the blood is carried by the bronchial arteries. This is in accord with the general plan of distribution of arterial blood to the lungs, for the bronchial arteries supply the bronchi and the pulmonary arteries the respiratory portion.

The lymphatic apparatus consists of a system of lymphatic channels or vessels and of lymphoid tissue which is intercalcated in certain situations along their course. There are two groups of lymphatic vessels, the superficial and the deep. The superficial lymphatic vessels lie in the subpleural connective tissue and drain into the broncho-pulmonary lymph nodes, located, in relationship to the larger bronchi, within the lung substance near the root. The deep lymphatic vessels are found in the walls of the bronchi and the blood vessels and in the trabeculae. These channels drain into the tracheo-bronchial lymph nodes at the bifurcation of the trachea. It is generally agreed that there are no lymphatic vessels in the alveolar walls. In addition to the lymph nodes at the root of the lungs, lymphoid follicles and minute masses of lymphoid tissue are found in the deep substance of the lungs. The lymphoid follicles are present, in relation to the large and medium-sized bronchi, especially where they branch. The minute masses of lymphoid tissue are found in relationship to some part of the primary unit, to the subpleural connective tissue and occasionally to the trabeculae. In relationship to the pleura, lymphoid masses may be found beneath the walls. A common site, under the pleura, is at the point where a trabecula joins with the subpleural connective tissue.

Most investigators are of the opinion that lymphoid tissue does occur in the walls of the blood vessels. Strahan and Simon do not confirm this, as they state that "the lymphoid mass lies in the wall of the bronchiole and extends through the whole of the wall up to the basement membrane of the mucosa."

A function of the lymphatic apparatus is to deal with particulate matter. A corresponding function of the blood vascular system is to deal with noxious substance in solution. Hence a lymphatic apparatus is necessary in the case of air passages and their terminations, which are constantly exposed to the influence of dust, but is not required in the case of the blood vascular system.

In considering the nerves of the lungs, the pulmonary plexuses at the root of the lung are formed by branches of the vagus and form the thoracic sympathetic ganglia. They give off fibers which form plexuses on the vessels and air tubes entering the lung. Ganglionic cells are found only in the larger bronchi. The bronchoconstrictor fibers are from the vagus nerve, while the bronchodilator fibers are from the sympathetic and arise mainly from the inferior cervical and first thoracic ganglia. The pulmonary vessels are supplied with both sympathetic and parasympathetic nerve fibers. Their effect on these vessels is not clear. The sympathetic fibers act as vasoconstrictors for the bronchial arteries.

The alveolar ducts give off alveolar sacs and alveoli. The alveolar walls of the ducts contain reticular fibers and elastic fibers and capillaries. The remainder of the wall of the alveolar duct between the mouths of the alveolar sacs, consists of densely packed elastic and collagenous fibers and some smooth muscle cells. The fibers and muscles rub between the mouth of the alveoli and pursue a wavy tangenital course between them. The mouths of the alveolar sacs are completely surrounded by a heavy, wavy wreath of collagenous fibers and continue from one sac to another and help to give thickness to the alveolar ducts. Thin straight elastic fibers accompany the collagenous fibers. The dense networks of reticular fibers which spread over the walls of the alveolar sacs are frequently continuations of these collagenous fibers. The latter in turn are connected with the collagenous fibers in the wall of the arteries, veins and bronchioles.

The "septal" cells which are found in the alveolar walls have the typical appearance and functions of macrophages or histiocytes originating from cuboidal epithelium. Macrophages or alveolar phagocytes can be found in the alveoli and contain varying amounts of black granules called dust cells, which originate from "septal cells" or alveolar epithelium and act in the "defense" of the lungs by removing dust particles.

In completing the discussion of the anatomical structure of the lungs, we mention the pleura, which is a serous membrane lining the pleural cavities and is reflected over the lungs as the

visceral pleura. It consists of a thin layer of collagenous tissue containing some fibroblasts and histiocytes, and several prominent layers of elastic fibers running at various angles to the outer surface. It is covered by a layer of mesothelial cells, morphologically and functionally similar to those of the peritoneum. A similar layer found in the thoracic cavity is called the parietal pleura. A prominent feature of the pleura is the great number of blood capillaries and lymphatic vessels distributed in it. The nerves of the parietal pleura are connected with the phrenic nerve and intercostal nerves, while the nerves of the visceral pleura are believed to be branches of the vagus and sympathetic nerves supplying the bronchi.

THE reaction of the lungs to dust particles may be either mechanical or chemical. The anatomical structure of the air passage tends to protect the lungs by the action of the ciliated epithelium, which has a tendency to sweep out any foreign matter, by the action of the musculature in the walls of the air passages, and by the part played by the alveolar phagocytes and the lymphatic apparatus. If the particles reach the alveoli, the cells lining the alveoli are stimulated to activity. These alveolar epithelial cells pick up the dust and, by phagocytic action, gradually penetrate into the pulmonary parenchyma. The dust then enters the intercellular spaces in the alveoli and is carried with the lymph to the peribronchial and perivascular lymph glands and finally is trapped in the lymph nodes at the root of the lung where fibrosis is early evidenced.

Some authors believe that the dust penetrates the smallest bronchi instead of the alveoli, with great accumulations of dust to be found in the interlobular and still more in the peribronchial and perivascular tissue outside of the cells. It has been noted that occasionally the dust penetrates these filters and then we find a metastases of the dust in remote organs, as the liver, spleen, kidneys, and bone marrow.

Dust may be deposited in the lungs only between inspiration and expiration, and the concentration of dust is greatest in the central area of the lung field which is subject to the largest respiratory changes of volume and to the strongest ventilation. and the circulation of the lymph fluid is most active in this area, which secures the quick resorption of the alveolar content.

The mechanical action of the inhaled silicon particles upon the lung, due to the sharp edges of the minute crystals, has been the significant cause of Silicosis, but recently the chemical action of the silicon particles upon the lung tissue has gained considerable attention and the most recent investigators are discounting the propensity of the mechanical action. They assume that a colloidal silicon is formed in the slightly alkaline medium of the tissues with the exposed surface of the millions of tiny crystals involving processes among which are absorption, surface tension, electric charge, and the giving off of molecules.

McNally states that, in his belief, "the damage that is done to the lung is due to the action of the fluorides in the blood attacking the silicon molecule and the soluble silicon tetrafluoride causing fibrotic changes."

There is considerable experimental data which favors the chemical theory of action. Gardner and Cummings have shown that the rate of reaction to particulate silica injected intravenously is inversely proportional to the size of the particles. Particles less than one micron in diameter act quite like colloidal silica and cause acute inflammation and even death from necrosis of the liver within a few weeks. Particles from one to three microns in size produce progressive fibrosis in the liver, with the formation of nodular lesions similar to those in the lungs caused by inhalation of silica dust. Particles from 10 to 12 microns in diameter stimulate the formation of small foreign body tubercles which do not change materially during a period of two years. Their control injections of the same quantity of aluminum oxide (emery) particles caused no necrosis or proliferation of the connective tissues and, according to Gardner and Cummings, responses of this nature clearly indicate that the reaction to silica is chemical in nature.

However, further study is very timely because there are, it seems, various factors involved which bring about the extensive pulmonary fibrosis not all of which are chemical in nature as shown by the fact that a high incidence of silicosis may appear in one foundry and be a very minor problem in another foundry in the same area with both having almost identical silicon content of the air.

THE length of exposure to silica dust necessary to cause silicosis varies, with the condition of exposure, from a few months to several years. MacDonald, Piggot, and Gilder were among the first to use the term "acute silicosis" in a publication describing silicosis in young women employed for four and a quarter years or less in packing soap powder. They were said to have been exposed to excessive concentration of silica dust, although no counts of atmospheric concentration had been made. However, the authors were more impressed by the fact that there were free alkali dust in the air of the working places (approximately 25% of such powders are alkalies) than with the high silica concentration. They concluded that since silica is soluable in an alkaline medium, the inhaled material would quickly dissolve to form colloidal silica in the moist lung tissues and, as a result, the connective tissue would respond by proliferation much more rapidly than is said to be the case when pure silica is slowly dissolved by the slightly alkaline body fluids.

In general, silicosis develops very slowly and it is only after 10 or 12 years exposure that there are marked clinical changes. Factors which seem to favor the acute development of silicosis are a high concentration of very fine silica particles with long daily exposure, the presence of free alkali in association with silica dust, the presence of infection, and the chemical action on the body fluids. Collis has stated that, "dusts appear more injurious as their chemical composition differs from that of the human body, or from elements of which the body is normally composed."

SILICON dioxide is a common constituent of all of our food, both of animal or vegetable origin, as well as being present in the air and drinking water. Lungs of individuals whose occupations do not especially expose them to dust inhalation contain increasing amounts of silicates in direct proportion to age. The silicates constitute from

3.5 to 10% of the total ash of the lungs. McNally reports that upon quantitative examinations of fetal lungs there is 0.11 mg. silicon dioxide per gram of dried tissue, showing that silicon dioxide begins to accumulate ever before birth. Dwellers of Pittsburgh were found to have from 1.2 to 5.3 grams of free carbon in each lung as contrasted with dwellers of Ann Arbor, Michigan, who had 0.145 and 0.405 grams. McNally, in discussing the silicon dioxide content of lungs in health and disease, upon analyses of 21 samples of lungs, gave an average of 1.13 milligrams per gram of dried lung tissue. Two cases which came to me at autopsy and upon which I submitted samples of the lungs for chemical analysis contained 1.78 milligrams and 1.57 milligrams, respectively, of silica per gram of dry lung tissue. Both cases had been Chicago residents for many years, the former a housewife and the latter an office worker. McNally states that any lung contining over two milligrams of silicon dicxide per gram of dried tissue indicates undue exposure to a dusty atmosphere.

Dr. Clayton Smith analyzed portions of lungs of tunnel workers for silica, and the percentage of silica in the dried lung varied between 0.84 and 3.1. It was lowest in a case with no silicotic nodules and the amount was not proportional to the period of employment. The highest figure occurred in a man with three or four years' previout employment in a coal mine, but another coal miner who worked seven years at the same occupation showed less silica than most of the men exposed only in the tunnel. Dr. Louis Gershenfeld gave analyses on sand pulverizers which showed from 1.8 to 2.5% of silica in the dry lung tissue. The duration of the employment, however, was questionable. MacCrae's percentages for ordinary silicosis in South African gold mines, states that the percent of silica in six dried lungs varied from 1.39 to 4.47 with an average of 2.63. He did not state the duration of exposure. None of these cases came to autopsy at the end of the period of employment, but observations were made nine to 20 months later when the men died of infection. Within that period some change in the lesions would be expected, particularly in the presence of infection. The significant feature is that the gross anatomical changes which had developed at the time of death were not sufficiently extensive of characteristic to be recognized as silicotic in origin. This tends to establish the fact, in the opinion of MacCrae, that silicosis can develop under short periods of employment with high concentrations of exposure but the reaction is of microscopic proportions. They were all young so they could not have worked long.

Among miners of Broken Hill, Australia, where the stone contains an average of 12.23% of free silica and 44.57% of silicates, the men develop a fibrosis of a different type from that due to free silica. The lung tissue is not as dense as that of men suffering from silicosis caused by free silica. The x-ray findings are also different and are described as soft.

It is generally assumed that only the quartz of free silica content of granite dust is of significance in the production of disease, that the other components act as inert diluents which reduce the concentration of silica when inhaled, and that because of such dilution, granite pneumoconiosis develops much more slowly htan that produced by pure silica. On the other hand, it is generally possible to assume that the non-silicous components of granite dust may of themselves excite biologic reaction and that possibly they may modify the characteristic effects of pure quartz. Granite pneumoconiosis does not begin with the formation of nodules in the lymphoid tissues as in ordinary silicosis, but it presents only the characteristic perilymphatic proliferation of the non-silicous dusts. It does not create a condition of increased susceptibility to tuberculosis as quickly as that produced by pure silica.

Asbestos is found in the lungs in two different forms, namely as black and opaque particles and as asbestosis bodies which average about 0.075 mm. long and are composed of small discs with rounded margins arranged in the form of a tapering rod with a bulbous extremity. They are golden yellow or brown in color and give a distinct iron reaction. In lung sections they are often seen surrounded by foreign body giant cells. The asbestosis bodies result from changes which the asbestos fibres undergo in the tissues. The soluble parts of the fibres are removed by the tissue fluids and a delicate frame of silicon remains which absorbs proteins. Thus, the needle-shaped frame becomes surrounded by a mantle of proteins which coagulate and into which the silica is dispersed. This initiates the disintegration of the frame which is finally resorbed, according to Berger.

Thus, if other injurious dusts besides silica are inhaled over a long period, there is a diffuse cellular fibrosis occurring in the walls of the bronchioles, vestibules and atria. The fibrosis involves the supporting connective tissue of the adjacent blood vessels and the walls of the alveoli in the neighborhood to some extent. There is usually desquamation of the epithelial lining of the bronchioles and vestibules, often without evidence of a definite exudate. In very advanced cases of the condition the fibrosis may extend to the septa, to the supporting tissues of the bronchi and larger blood vessels, and to the alveolar walls. Here even the greatest amount of fibrosis is localized to the supporting tissue about the entrance of the primary unit. It may advance to such a degree that the bronchioles become constricted, and sometimes may be reduced to mere slit-like openings. Rounded nodules of cellular character are sometimes seen, the dense hyaline type of nodule is usually absent. Dust containing only a small percentage of silica may also give rise to these changes, and there is a greater liability to generalized fibrosis when a low grade type of infection (not necessarily tuberculosis) complicates the dust effect. The changes above appear to be primary lesions caused by the inhalation of any injurious dust, but when the great majority of the inhaled particles are silica, there develops in addition a specific and localized type of fibrosis—the silicotic islet.

Thus when the particulate matter which enters the pulmonary alveoli is taken up by the phagocytic cells of the alveolar septa the cells enlarge in size, are injured by the foreign matter and lose their action and disintegrate. Through this breaking down process, the silica-laden cells gradually progress to the pulmonary lymph vessels where they coalesce and block the lymph flow, thus stimulating connective tissue growth about the bronchi

and blood vessels. Some of the silicon particles are removed by the local histocytes and are carried into the surrounding tissue. The majority of the silica particles are then filtered out in the small lymph nodules along the bronchi and underneath the pleura. Any subsequent inhaled dust now remains in the alveolar wall because by the blocking of the lymph vessels its removal is prevented.

Solid, deep black and firm areas are finally found which occupy the mid-portions of the lung, sparing the apical parts and the base, which become emphysematous. According to Ohr, this emphysema may be so marked that by rupture of one of the blebs a spontaneous pneumothorax develops. In the center of the diffusely consolidated areas which cut like wood, one often finds small cavities which are surrounded by friable tissue and are filled with a thick black inky fluid. These cavities are from one-half to one and one-half centimeters in diameter. According to a number of authorities, these cavities are due to a non-infectious, autolytic liquefaction of the poorly vascularized scar tissue. In addition to liquefaction, the scar tissue may undergo calcification and eventually ossification.

The cardiac embarrassment due to the fibrosis causing obliterating changes in the branches of the pulmonary artery, interferes greatly with the pulmonary circulation. Due to the lack of circulation to the right ventricle of the heart, the wall becomes thickened to from 6 mm. to 10 mm. as compared with a normal thickness of from 3 mm. to 4 mm., and the decompensation and failure of the right heart is a common cause of death in advanced silicosis.

The formation of a silicotic islet of non-infective type consists of a central mass of dense hyaline fibrosis which are arranged in whorls. This process is brought about by the silican-laden cells piling up and becoming surrounded by spindle-shaped cells which enter these clumps and produce a collagenous ground substance. Layer after layer of concentrical fibrils is laid down about and between the silica particles, the fibers fuse to coarser trabeculae which become hyalinized and a sclerosed granuloma results which gives the characteristic appearance of whorls. This is surrounded by a comparatively narrow zone of cellular fibrosis laid down in concentric laminae. Scattered through this mass, lying between the connective tissue cells and fibrils, there are scanty large and small round cells and a little particulate matter. In suitably stained sections, a deposit of fat may be seen—the degree varying with the age of the nodule. The early fibrotic nodule thus formed is surrounded by numerous dust cells. The growth of the silicotic islet continues by successive new arrivals of dust cells and subsequent extension of the fibrosis at the periphery of the fibrotic nodule. The periphery of the nodules contains much more silicon than the center.

The composite islet is formed by the coalescence of two or more single islets, each of which has developed in relationship to some part of a given primary unit. It is seen grossly as a single islet and recognized only microscopically. The growing islet, especially if composite, displaces and distorts the alveolar walls and blood vessels and the terminal respiratory passages; some or all of these structures may be incorporated in a composite nodule. In a developed condition of silicosis, the silicotic islets are to be found distributed through the deep substance of the lungs and also just under the visceral pleura. In the deep substance it develops in the region of the entrance to the primary unit or lobule. It always develops at a site which is easily reached by dust cells which have phagocyted pigment and dust in the alveolar spaces.

Non-infective silicosis may appear as a "massive" type of fibrosis. Microscopically the lesions consist of numerous contiguous single and composite islets. The intervening alveolar tissue is much compressed and collapsed, but there is no evidence of inflammatory infiltration, breaking down, nor of any definite matting together of the individual islets. The larger blood vessels as a rule are not constricted, and the adjacent alveolar tissue shows no significant change.

Before the silicotic changes occur in the lungs, and subsequent to the appearance of some fibrosis in the lymph glands, changes as a rule take place in the pleura and sub-pleural tissues. These changes occur first at the sites where the lymphoid tissue is found in contact with the periphery of the primary units where the lesions within the lung develop. There is an increase in pigment seen grossly at first as fine mottling, then as coarse mottling or marbling and later as a diffuse pigmentation. With the development of the sub-pleural silicotic lesions the islets grow into the pleural tissue appearing to displace the pigment there, and ultimately reach the serous surface, where they show as pearly white impigmented spots surrounded by a ring of pigment. At this stage they may be just palpable on the surface of the pleura. As the islets increase in size they become flattened and present the appearance of small "plaques" which are raised above the serous surface of the pleura and are easily palpable. They are usually more numerous on the posterior aspect of the lungs, though they may be general, but they are rarely found in the inter-lobular pleura.

In simple silicosis there may be no other changes, but there may be areas of thickening and opacity, and adhesions due to local pleurisies which are common in the infective type. It is possible that one might find only one or two large plaques, not infrequently calcified and associated with the tuberculo-silicotic sub-pleural lesions. The number of sub-pleural plaques gives no indication of the severity of the lesions within the lung.

FOR a diagnosis of silicosis of the "simple" type, the lesions of the lung substance are the most important. To establish a diagnosis there must be present in the lung substance an appreciable amount of palpable fibrosis which is typically of a nodular character. When hyaline fibrosis appears within the developing islet, the latter becomes just palpable. With the increase in size the islets become visible as well as palpable and tend to project above the surface on cutting. They are pigmented black, and retain this black pigmentation to considerable size. They are usually discrete or they may be uniformly distributed. They sometimes do appear in the upper lobes in the sub-apical region and may be confined to this area, which suggests an associated tuberculosis. As the silicotic process develops, the islets increase in size to approximately five millimeters. They remain uniform in shape and color, black with sometimes a grayish tinge caused by the fatty degeneration.

There is a certain amount of bronchitis present, noted by the thickened walls and lessened secretion, and there is also some amount of emphysema present, especially marginal, which is a feature of the advanced stage of silicosis.

A case of moderately well marked silicosis of the simple type will show enlarged, pigmented, fibrosed root glands, moderately numerous subpleural plaques, and on section there will be numerous medium-sized islets of pigmentation diffusely scattered throughout the lung substance, the majority of which are palpable and project above the cut surface. There may be one or two areas of massive fibrosis, and bronchitis and some marginal emphsema may also be present.

TUBERCULOSIS is the most important complication of silicosis. It may arise from the latent focus within the lungs, especially from a lymph node, with hematogenous dissemination to the lungs, or it may be a new exogenous superinfection. It may occur before, simultaneously, or after the development of silicotic nodulation. The isolated "infective" islet consists, in the early stages, of a mass of very cellular fibrosis lying within an aggregation of dust cells. The fibrosis greatly simulates granulation tissue, but frequently blood vessels are not observed. Some collagen fibers are laid down, but usually before an appreciable degree of dense fibrosis occurs necrotic degeneration of varying degree takes place in the center of the islet, and this change becomes more marked as the condition advances. The tuberculosilicotic islet attains larger dimensions than islets of the non-infective type, and usually develops more rapidly. Evidence of inflammatory change is commonly seen also in the surrounding lung tissue in the form of increased vascularity, localized edema, cellular infiltration, catarrhal change, and later true inflammatory fibrosis. If it is extensive, an area of "infective massive fibrosis" is produced and similar changes occur in the lymph glands.

The "massive fibrosis" of tuberculo-silicotic type differs from that of non-infectious type in that the central portions of the nodules tend to show necrosis, the fibrous tissue growth in exuberant, and the islets are matted together by an inflammatory fibrosis which, in progressive cases, these lesions tend to spread more or less slowly. Large areas of lung may be occupied by this type of "infective fibrosis". On the other hand, the excessive fibrotic reaction may become preponderant, with the result that such lesions, whether "nodular" or "massive", may show long periods of practical arrest. In lesions of this type there may be no definite histological evidence of tuberculosis beyond the suggestive indication of necrotic degeneration of the central portions of the islets. It has been found in many instances, however, that material taken from these lesions produces tuberculosis in inoculated guinea pigs.

The superimposition of tuberculosis infection upon the silicotic nodule, probably by the blood stream, shows the appearance of the infection in the "dust cell" area at the periphery of the nodule. This applies to both the isolated and massive islets. Histological sections reveal tuberculosis granulation tissue within the "dust cell" area and in limited parts of the surrounding lung tissue. In the more advanced cases there may be marked caseation, and the picture may then show fibrotic islets embedded in areas of caseous tuberculosis. In some instances there may be little or no modification of the infective process; in others the caseous change becomes in turn limited by fibrosis. The above described lesions illustrate certain results of the reaction to tuberculosis infection in the silicotic lung, they are not purely silicotic nor purely tuberculous, and justify the term "tuberculo-silicosis". This term is not applied to the conjunction of silicosis with an unmodified tuberculosis in the lung where the tuberculosis occurs apart from the silicosis.

In an advanced case of tuberculo-silicosis, which shows no sign of overt of obvious tuberculosis, there will be enlarged root glands with periadenitis usually showing irregular grey or greyish-black pigmentation and fibrosis, sub-pleural plaques of irregular size, opacities and thickening of the pleura with irregular dense adhesions usually over the upper lobes, and on section the lung shows moderately numerous to numerous large irregular palpable islets with areas of irregular massive fibrosis in which points of necrosis may be apparent, the whole area being pigment grey to grey-black, and bronchitis and emphysema are also present.

GARDNER divides the pathological changes of Pneumonoconiosis into five general phases: (1) early diffuse parenchymatous disease due to the accumulation of phagocytes and local inflammatory changes in the immediately adjacent connective tissues; (2) linear perilymphatic proliferation; (3) beading of the trunks due to chronic proliferative changes in the associated lymphoid tissues; (4) enlargement of the mediastinal lymph nodes due to proliferation of the locat connective tissues; (5) late chronic proliferation and nodular formation in the finer connective tissue of the pulmonary parenchyma. These differences in reaction are due to the localization of the various types of particles effected through the activity of the phagocytes, by the degree of impairment of the lymphatic system, and by the capacity of the various kinds of particles to set up proliferation in the connective tissues.

In considering the clinical aspects of any disease, it must be considered in relation to its pathology. In silicosis the primary change is a slowly progressing interstitial peribronchial fibrosis which involves and eventually blocks the lymphatics and reticulo-endothelial system. As the fibrosis progresses the chest expansion and elasticity may be impaired, thus reducing its vital capacity.

According to Irvine, the practically constant clinical signs are a certain lack of elasticity of the chest wall during the movements of respiration, together with a somewhat reduced air entry and a characteristic alteration of the inspiratory mumur from the normal "vesicular" character to a higher pitched or "harshened", "thinned", and commonly somewhat shortened type. The expiratory murmur, although somewhat prolonged, remains fainter than the inspiratory. This type of breath sound is very characteristic, with some modifications, of silicosis in all its stages, and this clinical sign has also the significant character of more or less complete generalization. It is first noticeable at the anterior, lateral and basal regions. In the minority of cases, however, the

breath sounds may be simply diminished but breath sounds which are simply diminished or which on the other hand, are merely somewhat louder of more pronounced than normal are not specially characteristic of silicosis. Usually there are no accomplishments, but a stray rhonchus may be heard here and there.

Other clinical symptoms which might be mentioned here which have some bearing are dyspnoea, especially upon exertion, which varies from slight to extreme depending upon the degree of involvement and which causes loss of strength; chest pain; cough; frequent colds; loss of appetite; constipation; flatulency; epigastric pain after eating; vomiting; posticus paralysis is frequent in advanced cases and is caused by adhesions between the indurated lymph glands and nerve trunk; haemoptysis is found in the advanced cases and increases when complicated with tuberculosis; in rare cases there may be a paralysis of the vocal cords with symptoms of tachycardia; compression of the trachea or bronchi may also occur; and there may be an induration of the glands causing compression of the pulmonary artery, which may produce a systolic of parasystolic mumur. While the cardinal symptom of silicosis is shortness of breath, the cardinal sign decreased chest expansion, and the earliest specific indication the characteristic roentogenographic appearance, the diagnosis should be made only by a correlation of all the facts, including symptoms, signs, occupational history, and laboratory and roentgen observations.

An individual with silicosis usually appears well nourished and apparently healthly, and except in advanced cases there is usually no evidence on casual examination of any impaired physical disability. As stated, very early in silicosis a limitation is developed in the expansibility of the chest and the chest continues to decrease as the condition progresses. In advanced cases the chest walls are apparently fixed and the patient moves only the abdomen and perhaps the lower part of the ribs in breathing. Gardner found in some of his cases that the maximum chest expansion was two centimeters. The restriction is symetrical.

The dyspnoea in the early stages is noticed only upon exertion. It increases in direct proportion to the period of exposure to dust. The fibrosis progresses gradually, until it completely fills the alveoli, and by the continuous displacement of normal functioning tissue produces a progressive dyspnoea. The pains in the chest are of a transient nature. The cough without expectoration is not of pulmonary origin but apparently from mechanical irritation in the upper respiratory tract.

Upon examination of the chest in the early cases there is a slight impairment of the resonance, and as the disease progresses this finding becomes more pronounced. The area of greatest impaired resonance is found over the hilus of each lung posteriorly, the impairment being evidently due to enlargement of the lymph glands of the hilus. In advanced silicosis there is indication of consolidation, and because of the great amount of fibrosis, the note obtained on percussion is quite dull. The impaired resonance is, then, a very constant finding. In the early cases of silicosis the tactile and fremitus are about normal, but as the disease progresses they diminish, because of the poor resonating quality of the lung tissues. This tendency is in contrast to the findings in regard to the localized fibrosis of tuberculosis, which gives an increase in these phenomena. In the early cases there is a general softening of the respiratory sounds throughout the chest, evidently attributable to the generalized fibrosis in the alveoli, while it is a localized fibrosis which alters the sounds in cases of tuberculosis. In the bases of the lungs, where vesicular breathing is normally found, the breath sounds are hardly audible.

IN CONSIDERING the x-ray studies as criteria of diagnosis, Pancoast and Pendergrass state that the stereoscopic roentgenograms, made in the postero-anterior direction are of the utmost importance, especially when unusual questions are to be settled. Fluoroscopic studies alone have no value whatever as medicolegal records. They are sometimes of value in following studies after x-ray but they do not give much information if the silica intake is unusually rapid as in some certain industries. The advantage is, however, in the determination of diaphragmatic excursions and costal expansion, which is a most important factor in estimating incapacity. In cases of rapidly progressive interstitial fibrosis, this observation may be the deciding factor in diagnosis or in the interpretation of roentgenographic appearance. It is also a means of determining just what additional roentgenograms may be required in a given case.

In advanced silicosis, the lungs are practically always emphysematous and the thicker lower portions contain much more than the normal amount of air and sometimes comparatively less interstitial pulmonary tissue, hence these portions are likely to be over-exposed, especially as to penetration, if the routine technic adapted to the normal individual of the same physical development is employed. It is therefore often advisable to make a set of roentgenograms by the usual technic for the build of the individual which will show the upper chest adequately but which may blot out much of the pathological change in the lower portion by over-penetration and then to supplement these roentgenograms by another set of lesser penetration to show to better advantage the changes in the lower lung fields. Often in advanced cases there are dense fibrotic consolidations, in the mid or subapical lung fields which may be tuberculous in addition to being silicotic. Another single roentgenogram or stereoscopic pair of very much over-penetrated exposures, especially if made with the use of the Potter-Buck diaphragm, may through additional light on these areas.

Pancoast and Pendergrass further state that lateral views are often very important in the location of lesions, in showing the posterior costophrenic spaces, in the determination of the exact degree of emphysema, in the estimation of cardiac size, and the condition of the heart and aorta, and sometimes in differential dignosis. A sagittal view does not give this correct information in advanced silicotic individuals. The marked emphysema of advanced silicotic individuals is likely to cause rotation of the heart and to diminish the width of the cardiac shadow in the sagittal view. The localization of the large dense areas of consolidation by the lateral view is often important for differential diagnosis. If these areas are found to be in the apex of the lower lobe they are not apt to be tuberculous, as the lower lobe apical tuberculous

lesions are not likely to take on this appearance.

The most important point in regard to the roentgenological aspect of pneumoconiosis would seem, then, to be the necessity of exact correlation of roentgenographic findings with pathological processes. Pathologically the dust particles must be 10 microns or less in order to be phagocyted, the average being two to five microns. Particles down to two microns may be detected by light field illumination, whereas those between one-half micron and two microns require the dark field method.

Pancoast and Pendergrass say that the dust cells apparently lodge in the outposts of lymphoid deposits. Some may reach only the most distal ones, others may go further; at any rate, they evidently initiate the change in chemical composition of the condition of the contained silica particles to a hydrated colloid, which is a destructive agent to cells. This change is supposedly brought about through the agency of the alkaline reaction of cells and body fluids. As a result, the dust-laden cells die and discharge their contents, which are destructive to the surrounding cells, in the lymphoid deposits. Other dust cells or phagocytes no doubt carry some of this discharged silica to some of the more central lymphoid deposits until eventually the pulmonary lymph nodes are reached. Wherever the altered silica is deposited there occurs a cellular reaction, with both infiltration and proliferation, and evenual local necrosis, usually microscopic in extent, followed by healing by fibrous tissue. The lymphoid deposits and pulmonary lymph nodes are thus involved in a progressive process. Pathologically these structures become enlarged and are the seats of subsequent fibrosis and both processes cause a lymph block in the perivascular and peribronchial lymph vessels. There is also proliferative thickening of the lymphatic walls which adds to the physical block or stasis.

At this stage the x-ray picture will show the enlarged and more or less fibrosed pulmonary density of the hilum shadows, while the lymph stasis and thickening of the vessels is likely to intensify the trunk shadows and linear markings. Also, the general enlargement of lymphoid deposits may cause a slightly increased density or faint haze of the portions of the lung fields most affected, that is, the middle fields and first on the right side. This appearance of prominent hilum shadows and increased prominence of trunk shadows and linear markings, with or without the faint haze, has been designated as the first stage of silicosis or pneumoconiosis. One may find this appearance in many other conditions, among which are chronic bronchial catarrh, bronchiectasis, passive congestion from cardiac decompensation, the infiltrating type of malignancy, and polycythemia, therefore this should not be accepted as an evidence of penumoconiosis unless all of these conditions are positively ruled out and the appearance has become present or more marked over a period during which serial examinations have been made. They further state that, even if it is due to pneumoconiosis, the appearance does not represent an incapacitating degree of the condition in any way, or most certainly not unless it has developed rather rapidly after starting in a dusty occupation capable of producing it. With very rapid development of the disease, one is more apt to find evidences of the interstitial type of fibrosis.

They (Pancoast and Pendergrass) state, moreover, that in cases in which silicosis is moderately slow in progress, the microscopic nodular lymphoid deposit proliferations tend to become conglomerate and to produce a macroscopic nodular process which appears on the x-ray as the typical rounded shadows from one to five millimeters in diameter, or even larger, and which are so characteristic of the conditions in many industries. This, which is designated as the second stage of the condition, is conspicuously absent in many industries, notably the granite cutter, sand-blaster, sandstone and asbestos workers, or those who are developing silicosis rapidly. This stage is termed by some authors as the nodular type, or preponderance of silicotic fibrosis.

Irvine says that at this stage, which he terms "the moderate degree of uncomplicated silicosis", there is sub-pleural and intrapulmonary nodulation, the nodules being more uniform and having a general distribution, moderately numerous and still "small" in size. There is often a slight patchy opacity of the overlying pleurae, with possibly local thickening, and the root glands show fibrosis to a varying degree but there is no significant amount of interstitial fibrosis and the alveolar walls are not thickened.

A tuberculous process coincident with silicosis at this stage can readily be distinguished, providing the nodules are not large or too widespread beyond the central lung fields. When the nodules are large and more widely disseminated, the radiographic diagnosis of superimposed tuberculosis becomes far more difficult and may be impossible.

There is another type of silicotic appearance which is of rapid development and almost invariably progresses to the terminal stage with little or no appreciable nodular appearance. With an unusually heavy intake of silica there seems to be an abrupt blockage of the lymph channels, then with only the pleuralward pathways open for the lymphatic travel of dust cells, these too are soon closed. Gardner's theory is that under such circumstances dust cells can and do penetrate the lymph channel walls directly into the interalveolar connective tissue and set up changes productive of an intersititial cell proliferation and subsequent fibrosis.

The rather typical roentgenographic appearance is a fine and rather homogenous haze usually first seen in the right lung in the middle third and just below, and later in the left lung. It tends to spread rather evenly into larger areas. It may or may not have the nodular type of the disease associated with it, depending upon the rapidity of the process. The detection of this type requires x-rays of the best quality and a stereoscopic set in order to identify the appearance as that of an intra-pulmonary condition. Fluoroscopic observations are necessary to determine whether or not the underlying cause of the hazy appearance is responsible for restricted diaghragmatic excursion. It must be differentiated from a chronic interstitial lung change resulting from pneumonia or continued upper respiratory infection. A tuberculous spread from another lesion must also be ruled out.

The third stage or, as Pancoast and Pendergrass prefer to call it, "the terminal diffuse fibrosis", is almost always incapacitating to the fullest degree. The x-ray presents three striking characteristics;

a nodular type may become more and more conglomerate, with more or less interstitial fibrosis; an interstitial type may progress to a terminal condition and present appearances somewhat similar to, and sometimes almost indistinguishable from, those of chronic diffuse fibroid tuberculosis or the terminal fibrotic stage of a Friedlander pneumonia in a patient who has survived; and large consolidations may be found in the subapical region. These may be symmetrical as to size and similarly located, or they may be much larger on one side. Many of them no doubt possess a certain tuberculous element. Tuberculosis may be strongly suspected when these consolidated areas are peripherally or subpleurally located and especially if there is a homolateral deviation of the trachea. Cavity, of course, implies tuberculosis in almost every instance, whether the sputum is positive or not. Sometimes the presence of cavities may be determined only by over penetration of these areas.

Pulmonary asbestosis differs quite materially from practically all other phases of true silicosis. There is an appearance suggesting increased prominence of hilum and trunk shadows, especially in the early stages, but this is overshadowed later by other features. Sooner or later, there appears a rather homogeneous density in the lower lung fields, evidently due to an interstitial type of cell proliferation and fibrosis. Later on there is a further increase in this density in a wider field, with a fine network of fibrosis, which may be a transition of interstitial into a mild terminal fibrosis. There is very little nodular predominance and it is always inconspicuous. With this appearance diaphragmatic excursion is limited and the outline may be uneven. The diaphragmatic excursion in indistinct, the costophrenic angles are clouded, there is the absence of the typical emphysematous appearance of advanced silicosis, with the small shadow of the rotated heart, and the left cardiac border is often very indistinct. The absence of marked evidences of emphysema and all these other differences might be explained on the basis of a lack of aeration due to the very peculiar characteristic pathological features of asbestosis.

The predominant pathological feature is the presence of the asbestosis bodies. These bodies are of various sizes and are found in various stages of development in many localities, but particularly imbedded in fibrous tissue, most of them in large clumps in the alveoli and very large ones in the terminal air passages. Whether any fibers are sufficiently small to be phagocyted is uncertain. Certainly they do not reach the pulmonary lymph nodes. The bodies increase in size from the original fibers, probably by colloidal aggregates of blood protein, and contain an iron salt. It is possible that this reaction to the inhaled asbestos fiber is, after all, a safeguard against even more serious pulmonary changes.

Gardner has described the relations of the largest of these bodies to the terminal air passages in which they are found and states that they are apt to be surrounded by a fibrous collar in the air passage wall, and it has seemed quite likely that this collar surrounding what is really a foreign body of increasing size, together with the presence of large clumps of the smaller bodies in the alveoli, must be conductive to a condition of multiple minute areas of atelectasis, or drowned lung. Such a state of affairs might well explain the lack of x-ray evidences of emphysema and the other peculiarities of asbestosis as compared with silicosis, and also the intense dyspnoea and cyanosis of late pulmonary asbestosis.

IN THE use of polarized light for the diagnosis of silicosis, all transparent substances the visibility of particles of which is increased when the light which they transmit has passed through crossed Nicol prisms, possess the property of birefringence, or in other words, have two indices of refraction. These indices are known as the "ordinary" *(w)* and the "extraordinary" *(e)*. The amount of divergence between these two indices *(w-e* or *e-w)* is a measure of the strength of the double refraction (birefringence) of the substance; the greater the difference between the two indices, the more increased is the visibility of the particle in polarized light. Thus for rutile (one of the forms of crystallized dioxide of titanium and a strongly polarizing substance) (in Na light=2.616, and =2.903; the birefringence of this substance 2.903—2.616=.287) is therefore high, and very minute particles of this substance are therefore conspicuous in polarized light. For quartz, this mineral has a low power of double refraction (0.009), and small particles of it should not, therefore, be markedly visible in polarized light.

The degree of visibility of a birefringent particle, when viewed in polarized light, is also dependent upon the inclination of its crystallographic axis to the line of vision; if this be unfavorable, that is to say, if the crystallographic axis be parallel to either of the Nicols, the particles, though definitely birefringent, become quite invisible. The optimum inclination of the axis of the particle to the axis of either Nicol is that of 45 degrees. The intensity of illumination is an important factor in the visibility of birefringent particles. The polarizing effect in histologic sections is more marked with artificial illumination in a dark room. Birefringent particles which are possessed on an intrinsic color (red or yellow) are more conspicious when daylight is used, than with those that are colorless.

The method for the identification of inorganic substances in tissue is a combination of petrographic and histological techniques. Paraffin sections are cut, removing the embedding medium with zylol and alcohol, drying the sections in air, and then removing the organic material by heating in a muffle furnace. There remains firmly sintered to the slide the inorganic constituents, both those normal to the tissues and any extraneous substances such as inhaled dust. These incinerated sections are examinted under polarized light with a microscope. The number and character of the particles is observed and recorded. Since all heat-resisting substances are thus visualized, the sections are treated with aqua regina for two to 18 hours which dissolves everything but silica, some of the silicates, titania and a few of the rare elements. The sections are dried and reexamined in the same manner. As a general rule, quartz shows only a moderate brilliancy under the polarizing scope whereas some of the fibrous silicates, like sericite, are much more brilliantly refractile. However, absolute identification of the particles must depend upon petrographic methods.

The appearance of the silicotic lung in polarized light under low magnification is suggestive of the starlit sky. Under higher power (eq. Zeiss obj. DD) the great majority of what previously appeared as tiny points and lines of light are resolved into bright elongated images of apparently very definite outline. A common form appears as a fine, irregular but usually sinuous line, or two or three such lines converging at their ends and suggestive of a small cluster of down-barbules. Some of the points of light will be resolved into small shapeless patches without definite boundaries, and others into minute groups of such patches, as though the translucence of a tiny window had been largely obscured by roughly smudging the glass. The increase in illumination, or in the degree of magnification, increases the apparent number of such elongated and shapeless objects. Rarely it is that two or more elongated images lie in exactly the same focus. Oftentimes the bright images seen do not represent true projections of the outlines of the objects but are optical illusions. In silicotic sections their polarized images are approximately their true image, their average sizes being from one-third micron to five and one-half microns.

IN DIFFERENTIAL diagnosis, the conditions which are most likely to be confused at the perivascular-peribronchial-lymph node stage are, according to Pancoast and Pendergrass, passive congestion of the lungs as a result of cardiac decompensation, passive congestion associated with coronary thrombosis, advanced bilateral bronchiectasis, asthma, infiltrating or permeating malignant metastases, polycythemia or erypthremia, mycotic infections. In the early interstitial stage, the confusing conditions may be a tubercul--ous pneumonic process, interstitial pneumonitis, bronchosinusitis, rheumatic pulmonitis. In the nodular predominance, the confusing conditions may be nodular metastatic malignant conditions of the lungs, actinomycosis, sporotrichosis, leptothrix infection. In the advanced diffuse or terminal fibrosis stage the confusing conditions may be chronic diffuse tuberculosis, or an accompanying tuberculosis which should be definitely diagnosed. Occasionally such areas may simulate the presence of a mediastinal tumor, carcinomatous pulmonary affections, and leutic pulmonary affections.

Among the complications are pneumonia, pleurisy with effusion, emphysema which directly produces the cardiac failure in cases where that condition develops, bronchiectasis which is rare, cancer in its relation here is ill-defined and it is thought at this time that penumonoconiosis is probably not a pre-disposing factor in primary bronchogenic carcinoma, and tuberculosis. In the relation of pneumoconiotic pulmonary changes to tuberculous infection, the primary pneumoconiotic pulmonary changes are to be considered as having a favorable influence on the later tuberculous infection, since they show that the organism has a tendency to answer by a cirrhotic reaction to pulmonary irritations. A secondarily appearing, hematogenous tuberculosis may be inhibited by the pre-existing indurative pneumoconiotic tissue in its propagation method, by constriction of the internal path of infection. An intracanalicular tuberculosis, which usually has a malignant course, is influenced neither favorably nor unfavorably by a pre-existing pneumoconiosis. Pneumoconiosis does not prevent the occurrence of a tuberculous infection. A pneumoconiotic injury affecting secondarily a pre-existing tuberculosis may influence the affection in the following ways: the irritation conditioned by the dust and consequent tendency to catarrhal affections will undoubtedly have an unfavorable influence on the course of the disease; and the pneumonconiosis may inhibit the hematogenous infection of tuberculosis, and may therefore have a favorable influence. Which of the two affects will predominate will depend on the various constitutional conditional factors, the structure of the dust, the amount of inhaled dust, the type of work the individual does, the duration of the injury, and the various protective mechanisms are the conditional factors and play an important role here. The virulence of the tubercle bacilli has an important influence on the pathological process. There are numerous constitutional factors which influence the course of the disease, such as congenital general and specific resistance, individual ability of the organism to react by connective tissue formation, structure and developmental possibilities of the chest and the tissues of the thorax. The following are also important conditional factors: hygiene, social condition, sources of infection, misuse of alcohol and tobacco, body hygiene, sport, and a number of psychic factors.

The malignant tuberculous forms are unfavorably influenced by a consequent pneumoconiosis. A simultaneous effect of dust and infection with living organisms (especially tubercle bacilli) gives rise to a malignant mixed form of the two diseases, which has been designated by Ickert as "tuberculo-coniosis" or "conio-tuberculosis".

The pneumoconiosis is only one of the many conditional factors which may have a definite influence on the course of pulmonary tuberculosis; its type of action and its intensity of action in each case can be properly evaluated only after all other correlative factors have been thoroughly considered. Uncomplicated silicosis seldom effects the upper lobes or the periphery of the lung, lesions occurring in these places are likely to be tuberculous in origin.

THE prognosis in silicosis is good in early silicosis if the patient can be removed from his dusty occupation and be placed in the open air, but as the disease progresses the prognosis becomes less favorable, principally because of the liability to tuberculous infection. Silicosis is a progressive disease but the damage due to dust alone would not produce serious disability, the most important factor being that of infection—tuberculosis. In 75% of deaths of cases of silicosis (Irvine) the terminal condition was tuberculosis with silicosis. Recovery in the third stage is of a small percentage, but open air treatment is beneficial in some cases. Recovery means only subsidence of the inflammatory changes and symptoms, and not restoration of lung tissue.

There is a difference of opinion in regard to the suggested treatment by the administration of a non-silicous dust—as coal dust. Middleton thinks that once enough silica has been inhaled to produce the nucleus of fibrosis, the resulting inhalation of coal dust into a lung in which the lymphatic drainage has been disorganized increases the fibrosis and produces disability. Mavrogordato has been

unable to secure experimental evidence indicating that the presence of coal dust in the lungs exercises a protective action against tuberculosis, but thinks that the simultaneous inhalation of coal dust and silica facilitates the exit of silica-laden cells from the lungs, although a lung previously invaded by coal dust has no especial ability to rid itself of silica.

THE following are a few of the methods of prevention of dust; use of water, exhaust draught applied at or near the point of origin of the dust, dust traps and masks with positive air pressure, ventilation, making dust counts, and miscellaneous other methods.

A silicotic person should be removed from the source of irritation causing silicosis and be placed in an occupation in the open air. When complicated with tuberculosis, a climate high and dry and warm is advisable. Medical treatment is symptomatic and prophylaxis is more important than actual cure.

Menzel states that diathermy has a very favorable influence. He is of the opinion that if 10 to 12 diathermic treatments fail to have favorable influence on the condition, the condition is not due to pneumoconiosis.

Howard Goodrich states that treatment is most unsatisfactory, and that even if a worker stops working in silicous dust without any symptoms, these may develop later on.

He is also of the opinion that once a man develops the symptoms, stopping work will not prevent unfavorable progress of symptoms and pathological changes.

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The History of Industrial Medicine and Occupational Diseases

—An Historical Outline of Occupational Diseases, Industrial Hygiene and Surgery, Traced from Early Antiquity to the Great Depression of the Twentieth Century—

by

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Industrial Injuries and Diseases Due to Physical Agents

IT IS said that Thales of Miletus (640 B.C.) had first observed magnetic phenomena by noting that iron was attracted by lodestone. Theophrastus in 321 B.C. and Pliny in 7 A.D. made the same observation. The ancients were familiar with magnetism. The Chinese used the magnetic compass in 400 A.D. Peter Perigrinus was the discoverer of the magnetic needle.

William Gilbert, the physician of Queen Elizabeth, in 1600 began the scientific study of magnetism. The Leyden jar was discovered in 1745. Benjamin Franklin, of Philadelphia, in 1750 by the use of a kite in a thunderstorm drew sparks from the clouds and charged a Leyden jar.

Scientific electricity began with H. C. Oersted, the Dane, in the nineteenth century. A. M. Ampere, of France, in 1819-1920 showed that the electric current carried by a conductor produces magnetic effects. In 1831 Michael Faraday, of England, gave the law of electrolysis and contributed the outstanding discovery that whenever a magnet is moved near a coil of wire a temporary current is produced in the coil. When the principles of this discovery were applied, the outcome was the modern electric industry. The dynamo, telegraph, and telephones are examples of the application of Faraday's scientific achievement.

William Crookes (1832-1919) discovered thallium in 1859. His researches on evacuated tubes led to the phenomena produced by the discharge of electricity through highly exhausted tubes—the so-called Crookes tubes. In 1913 the Coolidge tube was invented.

Thomas Edison invented the electric light in 1878. Samuel B. Morse, of New York, created the telegraph in 1832, and Alexander G. Bell, the American physicist, invented the telephone in 1875. Nothnagel's views on electric shock were published in *The Lancet* in 1880. In 1895 W. C. Roentgen discovered the x-ray.

Many researches have been made in electrical shock. J. L. Prevost and F. Battelli, of Geneva, found that in alternating currents of low tensions, the heart was thrown into a condition of fibrillary tremor. With a tension of from 20 to 40 volts the heart became paralyzed but respiration continued. Simeon Buell, of Sheffield, England, showed that in electric welding the heat may rise to 7,000 degrees Fahrenheit and the luminosity may equal 8,000 candle power. He is of the opinion that the effects of the light are confined to the conjunctival and corneal surfaces of the eye. Should temporary blindness occur, it is due to vascular changes in disc and retina. He advises a helmet with a window made up of blue and red glass as a protective measure.

Lamotte Breuil, of France, showed that where electrolysis is used in the manufacture of calcium chloride the workers suffered from an acne due to chlorine compounds. D'Arsonval's studies in 1887 on the pathology and phenomenon of death apparently due to electric shock are classic. His method of treatment was that of artificial respiration.

Sir Thomas Oliver, of England, in 1898 experimented on dogs to find out how death was produced by electric shock. He demonstrated that respiratory failure follows after the heart action has ceased.

A. M. Bleile, of Ohio State University, in 1898 concluded that electric shock produces a constriction of the arteries, thus impeding the flow of blood which the heart is unable to overcome.

Grange, of England, in 1904 attributed the cause of death to haemorrhage in the medula oblongata, which, by pressing on the vagus, induces respiratory and cardiac failure.

M. Grizellitzer, of Berlin, in 1906 described electrical opthalmias among workers fusing steel rails by electrical currents.

Dr. Alfred C. Jordan, roentgenologist at Guys Hospital in London, in 1907 showed that x-ray workers become sterile (azoospermia).

Sir Thomas Oliver, of England, described the various injuries caused by electricity at the Fifteenth International Congress on Hygiene and Demography, held in Washington D.C., 1912.

In 1912 the Commission on Resuscitation from Electric Shock, representing the American Medical Association, the National Electric Light Association, and the American Institute of Electrical Engineers, appointed certain men to develop artificial methods of respiration. They were W. B. Cannon, Y. Henderson, S. J. Meltzer, G. W. Crile, E. A. Spitzka, W. C. Eglin, A. E. Kennelly, and E. Thompson. E. A. Spitzka and H. E. Raschi

were of the opinion in 1912 that electrocution is due to the electrolytic properties of the current seeking paths of least resistance along the course of the blood vessels. F. Chauvet and E. R. Hayhurst, at Chicago, in 1912, found that animal experiments on counter shock failed to prove that falls in electric shock will revive the victim.

The two Germans, Gildemeister and Diegler, sand that heart failure is the cause of death from electric shock. Campbell, Argyll, and Hill in 1924 while discussing counter shock as a method of resuscitation following electrocution, said that it is not favored by the medical profession because death results from ventricular fibrillation.

C. R. Langworthy, of Johns Hopkins, in 1932 by subjecting rats to electric shocks (248 volts) showed animals suffered from paralysis of hind legs and signs of hyperirritability of the nervous system, but decreased in subsequent shocks and developed an immunity and could stand higher voltage (1000), showing that injury is greater with first shock. S. Jellinek, of Vienna, 1928, observed by necropsies on men who died of electric shock that oedema and swelling of the brain were demonstrated. He advises the use of spinal puncture in the apparent moribund. This same investigator pointed out the changes which affect striated muscle, the "muscular spirals" due to electric shock.

Occupational Disturbances from Compressed Air

COMPRESSED air illness, commonly known as Caisson's disease, was first described in 1841 by Triger, a French engineer who first employed caissons in 1839. Le Roy de Mericourt, of France, in 1868 published a first report on compressed air disease among sponge divers. Paul Bert, of Auxoire, one of Bernard's famous and favorite pupils, dealt with gases in the blood, caisson's disease, and the toxic effects of oxygen at high pressure in an article in *La Pression Barometrique* in 1878. In the same year he proposed the theory of gas forming bubbles which act as emboli, producing the symptoms of caisson's disease.

The medical air lock was first used for decompression by E. W. Moire in the Hudson River Tunnel, in 1904. Leonard Hill and M. Greenwood, of London, in 1906 maintained that men can submit to seven atmospheres or 100 pounds without any untoward effects, provided decompression be effected gradually. Dr. H. M. Vernon, of Oxford, found that at body temperature the fat of mammals probably dissolves five times as much nitrogen as water or blood plasma.

Sir Thomas Oliver and Alfred Parker, of England, carried out researches in compressed air using mice as subjects. They found that symptoms develop on compression.

Prof. J. S. Haldane, of England, in 1907 advocated the interrupted or "stage" method of decompression. Gilman Thompson and Frederick L. Keays, of New York, in 1909 studied the symptoms in detail and also investigated the methods of uniform compression. Leonard E. Hill, of London, in 1912 investigated compressed air illness as well as the general evils of bad atmospheric conditions.

R. R. Sayers, of the United States Bureau of Mines, and Professor J. H. Hildebrand, of the University of California, in 1925 showed the possibilities of the use of helium oxygen mixture for the mitigation of Caisson's disease. Helium has the advantage of being less soluble than nitrogen, it diffuses more rapidly in body tissues and fluids, and likewise is more rapid in elimination during decompression.

Occupational Disturbances Due to Heat

MINOR disturbances caused by workers' exposure to atmospheric and artificial heat of certain intensities may result in disturbances of a serious nature, terminating fatally. Conditions such as heat stroke, heat exhaustion, and heat cramps may be found among laborers exposed to the hot sun rays. Stokers, furnace men, asphalt workers and workers in deep mines are also subject to these dangers.

W. D. Halliburton and F. W. Mott, of London, in 1903 asserted that the physiochemical cause of death from hyper-pyrexia is due to the coagulation of cell globulin. J. S. Haldane in 1905 showed the relationship of high humidity to diminished heat elimination with the resultant increase body temperature. He observed symptoms such as marked increase in the pulse rate, throbbing in the head, dyspepsia, exhaustion, and discomfort. First to emphasize a condition of the muscles in heat cramps was D. L. Edsall, of Harvard University, in 1908. He believed that this change is probably in the nature of an acute degenerative process. Dufour in 1909 noted the increase in polymorphonuclear cells of the spinal fluid at first, and that later the mononuclear cells dominate.

(R. B. H. Gradwohl and E. Schisler, of the United States,) in 1914 reported an increase in the blood urea nitrogen, uric acid, and creatin in patients with heat stroke and hyperpyrexia. Two Americans, P. McKenzie and E. R. LeCount, in 1918 studied the pathological changes in persons dead from heat stroke. These changes were oedema of the brain, hyperemia of the brain and lungs, hyperplasia of the spleen, cloudy swelling of the liver, kidneys, and myocardium, petechias, and the diminished yellow material in the adrenal cortices.

K. G. Hearne, an English investigator, pointed out in 1919 that a patient with heat stroke may lapse into a coma with increased body temperature, if there is a cessation of sweating.

Occupational Disturbances and Altitudes

PERSONS engaged in occupations in high altitudes, such as mountain climbers, aviators, and balloonists, are prone to undergo certain adaptive changes to counteract the abnormality of a new environment. This results in certain symptoms known as mountain sickness.

The conquest of the air was made possible by Americans: S. P. Langley in 1896 launched a steam propelled air craft which flew 1000 feet, and Orville and Wilbur Wright, two brothers at Kitty Hawk, N. C., in 1903 first flew with a heavier-than-air flying machine. Through these persons the world was given the modern aeroplane and a new occupation—that of aviation.

The hazard of high altitudes had already been investigated. Glaisher and Croxwell, English meteorologists, in 1862 ascended in a balloon to an altitude of 26,000 feet. They gave an early account of the symptoms of failing vision, paralysis, loss of hearing, and unconsciousness. Tissandier, the French balloonist, in 1875 was the

sole survivor of a fatal balloon ascent. He also described accurately the symptoms experienced at the altitude of 28,000 feet. Paul Bert, of Paris, in 1878 furnished clear experimental proof that the abnormal symptoms experienced at high altitudes are due to imperfect aeration of the blood with oxygen.

The acapnial theory, which attributed many of the symptoms to a lack of carbon dioxide in the body, was developed in 1899 by the Italian physiologist, Mosso. Kronecker, of Vienna, in 1903 endeavored to explain mountain sickness as due to a disturbance in the pulmonary circulation induced by diminished atmospheric pressure. The effects of high altitudes upon the blood elements were investigated by G. T. Kamp, O. O. Stanley and E. R. Hayhurst on Pikes Peak in 1903.

Joseph Barcroft, of Ireland, author of an important memior on the respiratory function of the blood, in 1914 investigated oxygen consumption in all the tissues. From 1908 to 1914 he made experiments of fundamental importance on the effects of mountain sickness and oxygen difficiency.

Other contributors to the knowledge on this subject are T. H. Ravenhill, Col. E. F. Norton, of the Mt. Everest expedition in 1924, J. S. Haldane and Yandel Henderson on Pikes Peak, 1912.

Occupational Hazards from Miscellaneous Causes

HAZARDS from the use of the air hammer among stone cutters are principally pulmonary, circulatory, and nervous disorders.

E. E. Southard and H. C. Solomon, of Boston, in 1916 reported a case of pain and numbness of the hand, with slight anaesthesia. David L. Edsall, of Harvard University, and J. P. Leake, of the United States Health Service, investigated the physical effects of the use of the pneumatic hammer.

Industrial Physiology—Fatigue, Ventilation, Temperature, and Special Senses

THE term "industrial physiology" was coined by Professor F. S. Lee, of Columbia University. The subject has attracted much attention, especially in the field of research in industrial fatigue, to which we shall first give our attention.

Industrial Fatigue

DURING the Great War the Ministry of Munitions of England appointed a committee of scientists known as the Health of Munition Workers' Committee to study industrial health and efficiency. The committee investigated the causes of fatigue, output in relation to hours, day and night work, employment of women and juveniles, sickness and accidents, industrial diseases and psychology, Sunday labor, hours of work, ventilation, illumination, and welfare work. It rendered a final report in 1918. This extraordinary report, issued under chairman Sir George Newman, will remain a classic in industrial fatigue research.

In the United States the United States Public Health Service, in conjunction with the Committee on Industrial Fatigue of the Council of National Defense and the Committee on Fatigue in Industrial Pursuits of the National Research Council, after three years of intensive labor, published a bulletin in 1920, comparing an eight-hour plant with a ten hour plant. This work is comparable to the British production. In France the Marey Institute of Paris conducted like researches.

The first historical reference to fatigue prevention was the setting aside of the seventh day as a day of rest in Assyria 6,000 years ago. This was later incorporated by Moses into the Ten Commandments. Solon, the Athenian law-given of the sixth century B. C., originated labor day. He dedicated it to the Moon (Monday), and made it the second day of the first week in September. The emblem of the Knights of Labor, who created Labor Day in the United States in 1882, bears a quotation from Solon: "That is the most perfect government in which an injury to one is the concern of all."

The study of human labor and its mechanical and physiological conditions originated in France. Coulomb in 1785 made the first steps by coodinating the physical and biological sciences.

Sir James Paget (1814-1899), the great English surgeon, said that "Fatigue has a larger share in the promotion or transmission of disease than any single causal condition you can name." A. G. Finlaison in 1853 stated that "The real practical difference in the distribution of sickness seems to turn upon the amount of expenditure of physical force."

W. Weichardt in 1904 advanced the theory that the cause of fatigue is a toxine generated in the overtaxed organism, and that when these products are injected in a non-fatigued animal it produces fatigue. E. S. Brogardus, of the United States, stated that the relation of fatigue to accidents is a very direct one and that the mechanization of industry enhanced the nervous strain upon the workers and so produced fatigue.

The German Wolf-Eissner in 1906 was of the opinion that during athletic training there is produced an immunity to the toxine of fatigue. This investigator obtained toxine from fatigued animals and injected it into a non-fatigued animal, producing all the symptoms of fatigue. This appeared to corroborate Weichardt's theory.

A. Mossaglia, an Italian investigator, showed in 1906 that when a portion of the parathyroid of a dog has been removed, the production of fatigue is always followed by tetanic convulsions and albumen in the urine. Sir Thomas Grander, of England, found slight traces of albumen in the urine of young soldiers after a march.

Mossaglia made curve studies in fatigue with laboratory animals by the use of the ergograph. Frank B. Gilbreth's fatigue studies in bricklaying, New York, 1909, was an early attempt to conserve motion, increase production and efficiency. This investigator and his wife, Lillian M. Gilbreth, in 1911 and in 1916 published further researches in fatigue and motion.

J. H. Ryffel in 1909 showed that in extreme exertion there is a loss of lactic acid through excretion in the urine. F. W. Taylor of New York in 1911 published his PRINCIPLES OF SCIENTIFIC MANAGEMENT, a remarkable contribution in efficiency engineering, elimination of unnecessary motion of workers, and fatigue conservation. He was the creator of the "Taylor system."

Josephine Goldmark, of New York, in 1912 published a monumental work on fatigue and efficiency. Alex Lipschutz, of Bern, in 1915 corroborated further Weichardt's theory by injecting

blood of overworked dogs into the blood of others, causing fatigue. He also showed that sleep is the great restorative.

That lactic acid is a product of muscular contraction was shown in 1915 by W. M. Flether and F. G. Hopkins, of Great Britain, Dr. R. W. Lovett, of Boston, and E. G. Martin, of Palo Alto, California, in 1916 experimented with the spring balance and developed a test for measuring fatigue.

A. H. Ryan, of Tufts University, Boston, a member of the Amreican Committee on Industrial Fatigue, developed what is known as the Ryan Test for fatigue in 1917. He uses an instrument to stroke the skin and produce the vasomotor reflex to test fatigued workers.

In 1917 Janet M. Campbell, of London, made investigations on the physical condition of women workers in relation to fatigue.

A method of testing the strength of certain groups of selected muscles in a fatigue study was developed when E. G. Martin, of Stanford, devised the spring balance strength test in 1918.

Much progress in fatigue research was made in 1918. The British Industrial Fatigue Board established in London in that year, has issued many reports. P. S. Florence, of London, developed the first systematic attempt to standardize methods of investigating industrial fatigue. C. S. Myers, of London, stated that "fatigue and practice work in opposite directions, and the work curve obtained is the resultant of these factors." Thomas Loveday, of Durham, a member of the scientific committee on the Health of Munitions Workers, investigated shifts, breaks, spells, pauses, and holidays in relation to fatigue and efficiency.

"Speed of production is the essential factor in accident causation which can never be neglected," stated H. M. Vernon, of Oxford, in 1918. Its action depends especially on the degree of nervous and muscular coordination possessed by the worker, and the psychical conditions, such as alertness.

A. H. Ryan and P. S. Florence, of the United States Public Health Service, contributed studies on rhythm in industry. The British Ministry of Munitions in its report of 1918 stated that fatigue may spring from many courses, such as the maintained use of intelligence and observation, and with varying degrees of the muscular effort necessary in every kind of work. E. L. Scott claimed that in addition to lactic acid, other substances, such as carbon dioxide and acid potassium phosphate carrying hydrogen ions, are freed during muscular contraction and may be of importance in causing fatigue.

Florence P. Sargent, of New York, in 1918 used factory statictics in the investigations of industrial fatigue. A. B. Hastings discovered in 1919 that the urine of men engaged in manual labor is much more highly acid than that of men at rest. This is a sign of acidosis due to fatigue.

During 1919, A. H. Ryan together with Jordan and A. B. Yates, of the United States Public Health Service, investigated muscular tonus in relation to fatigue.

R. A. Spaeth, of John Hopkins University, published in May 1919 a scientific report on the problems of fatigue in the *Journal of Industrial Hygiene* and gives therein a most comprehensive bibliography on the subject.

Jules Amar, director of the Reasearch Laboratory of Industrial Labor at the Conservatoire National des Arts et Metiers, Paris, published his book THE HUMAN MOTOR in 1920. He has laid special emphasis on the need for the application of study to the general principles of mechanics and thermodynamics in relation to the human machine. This is an important method of reaserch in the physiological effects of labor, fatigue, measurements of energy, external environment, equilibrium and locomotion, and their relation to industrial labor.

Amar says in concluding, "We must never forget that the battle of life is essential to humanity. Let us, therefore, assure to the worker the largest possible yield for his labour, so that he can both live in comfort and security, and also give value to those who employ him. Let us also watch over his health and organize 'social hygiene' . . .

Josephine Goldmark, a special expert at the United States Public Health Service, in 1920 published the studies of the committee of industrial fatigue on the comparison of an eight hour plant and a ten hour plant.

The National Institute of Industrial Psychology of London, established in 1921, and directed by Dr. C. S. Myers, have rendered invaluable research in industrial fatigue. They have published a journal in this important field.

Ella H. Oppenheimer and R. A. Spaeth, of Johns Hopkins University, made studies with fatigued rats in 1922. They found that fatigue did not markedly alter the susceptibility of these animals to toxins or infectious agents. If anything there was a slight tendency to increased resistance to tetanus toxine given to fatigued rats.

Some interesting experiments were made on rabbits by G. H. Bailey, of Johns Hopkins University. After intra-nasal inoculation of fatigued rabbits with pneumococcus, type one possessed an increased susceptibility, a rapid severe systematic infection and a greatly increased mortality. The control animals, when infected, indicated a greater general resistance.

The fallacy of Weichardt's theory was shown by F. S. Lee of New York in 1923. He proved when non-fatigued animals were injected with nitrogenous products from a non-fatigued control animal, it produced what Weichardt called fatigue.

T. H. Bast and A. S. Loevenhart in 1927 conducted researches on effects of lack of sleep as a factor in nerve exhaustion in animals, showing that the symptomatology of exhaustion was a drop in temperature as collapse came, increased pulse rate and slow decreased respiratory rate. Pathology cellular changes occured in cord, chromatin material of the nuclei decreased.

In New York City in 1929 a Noise Abatement Commission was organized by the City Health Commissioner to study the noise problem, its effect on fatigue and the health of the worker.

Ventilation

IN 1862 MAX von PETTENHOFER, of Munich, gave the world the carbon dioxide test as a measure for bad ventilation. This test was used as a standard until Carl Flugge in 1905 showed that the bad effects of vitiated air are due, not to chemical properties or impurities, but to physical properties, especially heat and humidity. The German physiologist, Hermann, had laid the foundation for these conclusions as early as 1884. Benedict and Milner, of the Carnegie Institution, Washing-

ton, published their classical studies with the respiratory calorimeter, made in 1903 and 1904, fully substantiating Flugge and his co-workers.

Leonard E. Hill investigated compressed-air illness in 1912, as well as the general evils of bad atmospheric conditions, and made important contributions to the science of ventilation and lighting of workshops. Flugge's researches were confirmed, beginning in 1914, by the New York and the Chicago commissions on Ventilation, whose studies notably advanced the physiology and engineering of ventilation. In the last decade, the ventilation studies carried out at the Pittsburgh Experiment Station Service and the American Association of Heating and Ventilating Engineers (Yagaglou, Houghton, McConnell, et al) developed the "effective temperature" and "comfort zone" standards. As a result of all of these investigations, modern mechanical ventilating exhausts and the substitution of dry by wet processes were greatly advanced for factories and mines.

For over 30 years, J. S. Haldane has done outstanding work in the study of atmospheric conditions and dust inhalation, and also in regard to miners' health and compressed-air hazards.

In the past decade, likewise, the great importance of body radiation as a means of effecting heat dissipation have come to the fore through the work of English investigators, especially Leonard Hill, A. Dufton, and H. M. Vernon, and in America by Aldrick of the Smithsonian Institution. H. M. Vernon's PRINCIPLES OF HEATING AND VENTILATING, 1934, summarizes this subject to-date.

Heating

WE HAVE already mentioned the Roman hypocaust of the early centuries A. D. The foundations for modern heating were laid with the invention of the Franklin stove in 1745, the development by Watt in 1765 of the steam radiator, and a system of hot air heating patented soon after by J. Green. The nineteenth century brought new inventions to this field. In 1831 Perkins devised high pressure hot water heating. Karl Bischoff introduced in 1839 the first successful gas heater. The climax in heating and stove development was the invention in 1855 of an atmospheric burner which mixed air and gas, by R. W. von Bunsen (the Bunsen burner).

Chimneys came into use in England in 1796, marking the beginning of ventilation for smoke-laden rooms where fires were used for heating interiors. J. R. Desaguiliers, a French physician, in 1723 attempted to ventilate the English Houses of Parliment after Sir Christopher Wren had failed. Since that time ventilation has steadily developed to its present day efficiency.

By whirling a heated ball on a string, J. Leslie of London in 1804 determined the variation in cooling caused by change in air velocity. W. Heberden of the English Royal Society in 1826 conceived the idea of determining the combined effect of evaporation, convection, and radiation, by observing the rate of fall of an ordinary thermometer previously heated to a high temperature.

To Leonard Hill, of England, belongs the credit for placing the subject of cooling on a quantitative basis in 1919. He also developed the Kata Thermometer, which measures the cooling power of the air, and the velocity of the air movement passing a given point irrespective of its direction.

R. R. Sayers, of the United States Public Health Service, in 1923 investigated the physiologic effects of high temperature and humidities with and without air movement.

The Departmental Committee of Great Britain on Factory and Workshop Ventilation recognized the removal of excessive heat and moisture as primary objectives in ventilation.

Through experimentation Professor Atzler, of Berlin, in 1924 determined the most rational methods of labor performances, especially tasks and problems on the physiology of work. The researches of F. B. Flin, of the United States Public Health Service, on the effects of high environmental temperature on the organism were published in 1925. R. R. Sayers and S. J. Davenport in 1927 reviewed the literature on the physiologic effects of abnormal temperatures and humidities.

Pneumonia has come to be recognized as a potent cause of death among heat-exposed workers (under cover), while the use of common salt in the prevention of heat stroke has been a real discovery.

Ventilation has now developed to the phase of "elective temperature" and "radiation balance," but with very little industrial application or even commercial recognition, while "air conditioning," is just knocking at industry's door.

The psychrometer, or U. S. Weather Bureau instrument, is a wet-and-dry bulb thermometer for measuring especially the relative humidity of the air. This, with the anemometer, pitott tubes, kata-thermometer and radiometer, have been developed in the present century to aid in the control of atmospheric conditions.

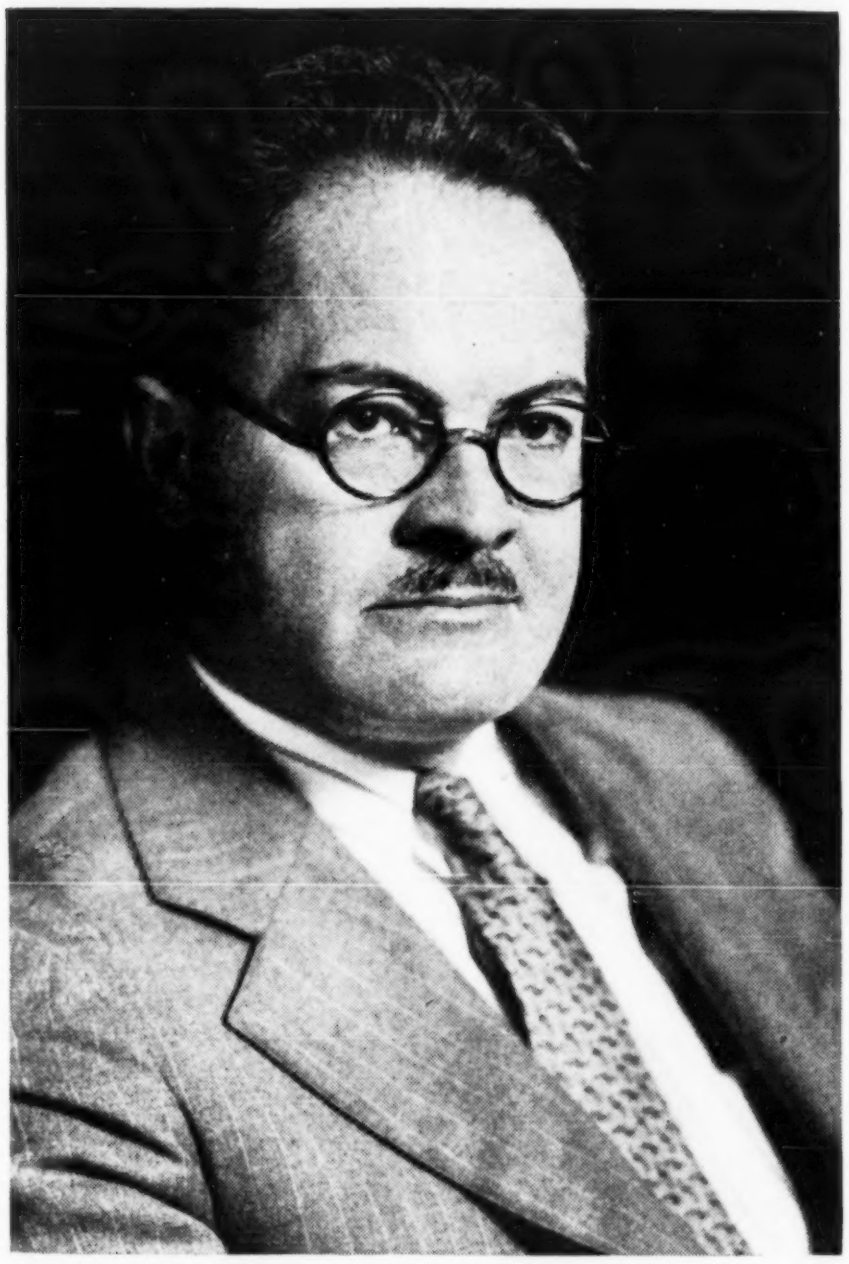
Special Senses: The Eye as Affected by Industry

FRANK SHUFFLEBOTHAM, of England, states that nystagmus is the commonest occupational disease known among coal miners, requiring from ten to twenty years to mature before causing complete incapacity. This disease was first reported among miners in England by Deconde in 1861, and after the compulsory introduction of the safety lamp in 1876 an increase in the number of cases began to attract attention. Hirschberg, a German, described in 1898 the characteristic symptoms, following an exposure of the eyes to the electric arc as "ophthalmia electrica." E. M. Alger reported the dangers of conjuctivitis as a result of hop picking. E. L. Collis, of the British Factory Inspection Service, found 28% of female sewing machine operatives had slight grades of nystagmus as result of fatigue of the eye muscles. Sir William Crookes proclaimed that glass-blowers' cataract is the result of the heat given off by the molten glass. T. M. Legge and K. W. Googby, 1912, regard the commonest industrial causes of disease of the retina to lead poisoning, holding that the sudden blindness is the result of the vascular change of the retina, either vaso-motor of hemorrhagic. A. A. Knapp in 1918 described toxic amblyophia due to carbon bisulphide intoxication. P. Lamson in 1924 noted in the pathological physiology in toxic ambloyopia observed in carbon tetrachloride intoxication that there was a lowering of blood sugar, which inspired A. S. Minot and J. T. Cutler in 1928 to put victims of such cases of poisoning on high calcium and dextrose diet. Cramer, of Germany, in 1907, attributed glass-blower's cataract to the chemical rays emitted by the blowing glass and calcium present in the sand.

Who's Who in I. M. & S.

—"I will study and get ready,
and maybe my chance will come"—

HERE is a man who knows what he wants. He has always known what he wanted. He knew it way back in the days when what he wanted was little more than "the vague circumference of the notion of an idea." And he knows now that he knew then—the record proves it; but how and why he knew then he doesn't know even now. Possibly, however, there *is* something in that phenonemon of destiny which, while it is preparing the circumstances a man must meet, is also—and elsewhere and independently—preparing the man to meet them.



CLARENCE OLDS SAPPINGTON

CLARENCE OLDS SAPPINGTON was born at Kansas City, Missouri, September 29, 1889. He came of Welsh ancestry, his immediate forebears moving over from Kentucky as a part of the mid-century infiltration of the middle west. He had finished the Kansas City grade schools and entered high school, when his family moved to Walla Walla, in the midst of the great wheat belt in southeastern Washington. There he completed his high school work and in 1907 entered Whitman College, where he carried a double major of biology-chemistry in addition to the classical courses which earned him his A. B. at graduation in 1911.

After college, he taught high school sciences in a small town in Idaho. He liked teaching, but the compelling urge toward medicine had begun to burn within him as early as his first year in high school, and this caused him soon to give it up, in order to begin his medical education. He did not recognize then that the scope and integrity of his ambition would demand, as the essential basis of its realization, a medical school of the very highest qualifications. That fact, however, was soon fully appreciated. Meanwhile, as is so often the case, aspiration was forced to compromise with necessity; and, finding a Chicago medical school where he could obtain enough teaching work to pay for his living and tuition, he enrolled in its courses. But almost immediately the vision of the far goal he had set for himself took in also the close actuality that, from where he was, he couldn't go fast enough under the restrictions of his then financial situation, nor far enough under the limitations of a "B" school. Finances having to come first, he got a job as a meter reader with a gas company in Boise, Ida. Then, his family moving to California, he went with them and got the same kind of job with the Pacific Gas & Electric Company—in the latter supplementing his income by doing private tutoring for students in dentistry who had not completed certain courses, mainly in the high school sciences, while preparing for special examinations.

In 1914 he was able to return to his chosen field, under circumstances which permitted him to concentrate his ability to learn in direct proportion to his desire to learn. He entered Stanford University School of Medicine in that year. Probably his first lesson there was that, although in his previous schooling he had acquired considerable knowledge, yet he had not understood the mechanics of study—and if there is any place where these must be mastered, it is a proper medical school. He mastered them quickly and permanently, and they are still the bright and shining instruments of his practice.

By special permission, during his years at Stanford, he held a place on the staff of the Livermore Sanitarium, a private institution for the treatment of mental and nervous diseases, situated not far from San Francisco. There he learned the fundamentals of the routine of handling nervous and psychiatric patients, and had also, in a laboratory which he set up, the opportunity for considerable animal experimentation—this enabling him to finish his thesis on "The Occurrence of Mitochondriated Red Cells in Experimental Anemias," which was later published in the *Archives of Internal Medicine* in 1918.

In his senior year at Stanford, a need for more interns at the San Francisco City and County Hospital, together with a little cooperation on the part of the Dean of the Medical School, made it possible for him to live at the Hospital and do an intern's work, in connection with regular class work, in the clinics during the afternoon. Graduating M.D. from Stanford in 1918—in the upper third of his class—he immediately took the State Board examinations in California, and shortly became Assistant Resident Physician at the Sacramento County Hospital, where a varied experience in emergency surgery crystalized the

technique of the ward and the operating room with which he had been in close contact as intern. A year or so there was followed by a short period as Assistant Resident Physician at the State Prison at San Quentin, California. This was a period of unusual persons and unusual experiences. Among the latter were the use of spinal anesthesia in major operations, prosthetic work in nasal re-fracture, special treatment of morphine habituism, the first testicular transplants done on the Pacific Coast, and the ever-present opportunity to study the psychology and psychiatry of the prison inmates. This was in 1919. And it was in the same year that he became Chief Surgeon for the Pacific Coast Shipbuilding Company at a point about 80 miles north of San Francisco. In charge of the medical relationships of 3,600 men employed in shipbuilding on government contracts, he had an excellent opportunity to work out the technique of the industrial physical examination, the treatment of minor injuries, first aid in cases of minor illness, and the various fundamentals of industrial medicine and surgery, with also the opportunity to carry on a private practice among the people who lived in the small communities adjacent to the shipyards. A little while later he established his private office for the practice of medicine and surgery in San Francisco. In conjunction with this, there was work in the outpatient clinic at Stanford, in the Division of Obstetrics and Gynecology, where in 1919 and 1920 he was Alternating Chief of the Womens' Clinic. The definite period of his general practice covers approximately four years, 1918-1922.

Toward the end of 1920, he was commissioned Assistant Surgeon in the United States Public Health Service at San Francisco—at the headquarters of the 12th District, covering California, Arizona and Nevada—in charge of examinations of soldiers, sailors and marines suspected of having tuberculosis. The examinations included chest studies, fluoroscopic readings and x-ray interpretations—with extensive reviews and consultations in connection with the X-day Departments of the Stanford University Medical School and the University of California Medical School.

AT THIS time Sappington's medical experience, while intensive in quality, had not been very long in length. It had been long enough, however, to show him the difference between being dissatisfied and unsatisfied. Happy in his medical work though he was, his vision could not rest within the purview of a general practice; his mind reached out to that *and* more than that. Over and above the intimate and personal working life of the physician and surgeon, he saw the more remote and somewhat abstract working life of the scientist. And he wanted to be both, to live both lives concurrently and together, to be increasingly more the scientist without becoming less the physician and surgeon. This, it will be granted, was almost a maximum of medical ambition. Probably it could be done—but how? The experience in the work of the United States Public Health Service showed the way. Putting the matter into words, as well as could be, the way lay along the path of preventive medicine. Looking down that path, Sappington saw himself following it; and he started by getting everything he could find that had been published on the subject, and not only reading it, but applying to it as well his carefully-learned mechanics of study. He hadn't gone very far, however, before he realized that the wide road of public health was paralleled by the little sidewalk of industrial medicine. At that time it was a rather narrow sidewalk; there were numerous gaps in its length; it wasn't very well traveled. But he had been Chief Surgeon for 3,600 men; and he had reason to believe himself qualified from experience to bridge any gaps that might confront him. And so he did some thinking:

ANCIENT as history itself, the employment relation has a history of its own. It was always a contractual relation: the wage for its *quid pro quo* of labor, and vice versa. Coming to the colonies, it was impressed with the common law of England; and spreading and growing in the early United States it reached the end of the nineteenth century still a matter of an individual contract upon which the common law superimposed certain duties of the employer: proper instruction; a safe place to work; proper tools, etc. In the stress of competitive industrial development these duties became so much a matter of generalized application and legalized resistance, as to develop in many minds the idea, and the necessity, of their statutory superimposition.

The first definite development was in the subject of pensions, as the reward of long service and a means of security in old age. These, however, began with the volition of the employers; until many years later they were not statutory. Meanwhile the matter of injuries while at work had reached and passed the climacteric which yielded the enactment in this country of compensation statutes along the lines of those previously enacted and found satisfactory in Germany and England. Indicative of the attitude toward the need for such laws was the statement of an early proponent: "There is a steel town in Pennsylvania with 25,000 inhabitants and 26 undertakers."

At this time, therefore, compensation for injuries had been established; it was then about six years old, and was slowly reaching toward the universality of acceptance which it has now practically attained. It was necessary; and it was effective—the best that could be done; and the average mind would say that it was good. But the scientific analysis showed that if compensation were to be all, if the condition were to be accepted in terms of its treatment—then the situation was upside down. Whatever logic there might be in statutory compensation for injury was only the first premise. The second was the answer to the question, "Why the injury?" And the inevitable conclusion was prevention. And more than that, there was in the literature about poisons and other exposures already more than enough to sustain the far-sighted view that occupational diseases must some time follow the same course toward statutory recognition.

Prevention—that was the key word: in occupational *injuries*, as they had become the present problem; in *occupational diseases*, as they were certain to become the future problem. What was the relation to the medical profession? Surgery: for the injuries which occurred in spite of prevention; medicine: for the occupational diseases which, with the development of chemical adaptations, would be inevitable until the means of pre-

vention were understood and applied. But with this difference:

The place of the surgeon was *after* the injury. Preventive surgery—what could it be but engineering, involving mechanical matters beyond the scope of surgical anticipation? Nothing that a surgeon could do to or for an employee would of itself prevent his carelessness with a buzz saw; only the engineers' preventive measures, applied to the saw, would temper that risk. And the surgeon would not be called upon until after the hazard and the exposure to it, and the personal equation in between, had resulted in the injury. If there were two barrels on the gun, the medical profession fired but one of them.

With occupational diseases, however, the medical man held the gun himself, and fired both barrels. Here his place was both *before* and *after*. Here the hazard itself was medical; the physician would be the one to diagnose it; the risk would be tempered in terms of medical science; and the exposure, and its effects and its treatment—all three medical—were all three directly involved with what the physician could do, both to and for the employee, in the way of prevention.

Thus industrial medicine—as medicine, and preventive medicine—would be *medicine in the employment relation;* the practice of industrial medicine would be the practice of medicine in that area of industrial, or employer, responsibility which is super-imposed upon any and every aggregate, any and every group, of individual contracts of employment; the *industrial* physician and surgeon would come in contact with his patients as individuals in their double capacities of human beings per se *and* human beings in employment. Industrial medicine would be a *field* of practice—not a specialty, but, on the contrary, the application of all specialties to all conditions needing them because of the employment relation.

Thus went the thoughts of this man who was thinking. And they went further. In such an important and tremendous field there could be a *specialist*. That was what he wanted to be.

And so he set about the business of becoming what he wanted to be—for he realized that no man by mere virtue of any general attainment or degree can make himself a specialist without adding the *special* attainments, and sometimes degrees also, which entitle him to call himself a specialist, and which alone will command his recognition as such.

IT WAS to be a long road. It was to begin with the adventure of the little sidewalk of industrial medicine. Harvard University was then offering a course in industrial hygiene and industrial medicine, and it was the only University where a doctorate in public health in this field could be obtained. He moved the necessary influence to get a fellowship at the Harvard School of Public Health, and there, beginning in 1922, he had two rich years, full of study, observation, reflection, and the development of a real philosophy in industrial medicine. Finishing in 1924, he received his Dr.P.H., the first American to receive this degree in the field of industrial hygiene.

In the summer between the two years at Harvard, he made a study of the incidence of lead poisoning, arising from the use of lead-conducted domestic water supplies in Massachusetts. The industrial clinic of the Massachusetts General Hospital believed, from certain experience, that this use of lead was a definite source of lead poisoning cases. Through the cooperation of this clinic, the Massachusetts Department of Public Health, and the Statistical Division of the Metropolitan Life Insurance Company, Sappington toured the State, gathered and tested his samples, and later made chemical and microchemical analyses, doing the latter work in the laboratories at Harvard, where Aub and his associates were then preoccupied in the matters later the subjects of their well-known monograph on lead poisoning. Sappington's study, carried on by him through extensive examinations of persons using lead-conducted waters, disclosed that about 25% of the people who imbibed the water conducted in this way, showed lead poisoning. The whole experience was the subject of the article "Lead Poisoning from Lead-Piped Water Supplies," in the *Journal of Industrial Hygiene*, for September, 1928.

The second year at Harvard was a teaching fellowship, as Assistant in the Department of Vital Statistics. This led naturally, to the thought of making his thesis a statistical presentation, if possible, of an industrial medical subject. Fortune favored the issue; there became available a wealth of material concerning the sickness and accident experience of a public utility in Boston, comprising about 15,000 cases of absenteeism over a number of years. With and from this he wrote his thesis.[1]

This was in 1924. Industrial medicine was assuming the stature of a profession; and, under the capable hands of its pioneers, it was shaping toward the definite and substantial recognition which was to come within the next decade. Why not, then, on the way back to California, get acquainted with some of its leaders? In New York City he met Watson, of American Telephone & Telegraph, who already had 10 years of constructive excellence behind him in the highest manifestations of industrial medical service; and Denno, of Standard Oil, whose Medical Department was already extending the best in American industrial medicine, and its concomitants of hygiene, sanitation and preventive measures, into the far-flung importance of international enterprise. In Philadelphia he talked with Lorne Hastings, of the J. G. Brill Company; in Washington with officers of the Industrial Hygiene Division of the U. S. Public Health Service; in Pittsburgh with Yant, Harrington, and others, of the U. S. Bureau of Mines Experiment Station; in Buffalo with Hourigan, of the Larkin Company; in Cleveland with Swan of Halle Brothers; in Chicago with Crowder, of the Pullman Company; Hart Fisher, of the Chicago Rapid Transit, and Harry Mock, who not only was one of the pioneers of the new field, but also, three years previously, had written its textbook.[2] Gentlemen all, they received him on the ground of their mutual interests, and their work—done, doing, and planned—was an inspiration.

In California again, he became Medical Director for Montgomery Ward & Company, Western Division, with headquarters at Oakland. There he remained until 1928, when he went to Chicago, as Director of the Division of Industrial Health, of the National Safety Council.

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1. This was published, in briefed form, under the title "Five Years' Sickness and Accident Experience in the Edison Electric Illuminating Company of Boston," in *Journal of Industrial Hygiene*, July, 1924.

2. INDUSTRIAL MEDICINE AND SURGERY, 846 pp.; W. B. Saunders Company, Philadelphia, 1921.

The Montgomery Ward affiliation gave him the opportunity to carry out the academic and practical experience gained at Harvard. The results—of the experience, and its application—are amply reflected in his writings, and in his annual and periodic reports which are definitely statistical. During these four years he was active in the work of all of the local groups and organizations which manifested an interest in industrial medical matters: as Consultant on Industrial Medicine for the San Francisco Tuberculosis Association; Editor, on Industrial Medicine, of *California & Western Medicine;* Secretary of the Section on Industrial Medicine of the California Medical Association; and engaged in various ways with the work of the Industrial Relations Association of California, and the Public Health Center of Alameda County, at Oakland. Added to this was teaching work, for he received an appointment from Stanford University of Lecturer on Industrial Hygiene, the lectures being a regular part of the course of the senior medical students in preventive medicine and hygiene. Simultaneously a similar teaching position was held at the Medical School of the University of California.

There came various offers—from Pomona College, in southern California, a professorship in hygiene, and the position of college physician; from Columbia University, the position of Assistant Professor of Public Health in the DeLamar Institute of Public Health, to be responsible for the teaching of industrial hygiene, and also to be Director of the Vanderbilt Industrial Clinic, with consultation work to be done; from Stanford, the position of Associate Professor of Hygiene and Preventive Medicine, to be responsible for the examination of freshman students, the teaching of college hygiene in the various classes, and to work as a consultant with the athletic directors. Weighing these against the career in industrial medicine for which he had been preparing himself, he said, "although I would have enjoyed being on the faculty of a representative institution, and could probably have contributed something and gained much, I selected the group through which I could enter into the work of industrial medicine and hygiene."

HE BECAME Director of the Division of Industrial Health of the National Safetv Council, Chicago, in September, 1928. There he was consultant and advisor to many types of manufacturing, public service and other industrial and semi-industrial groups. This work had many ramifications. Realizing that dust problems were to play an important part in industrial medicine and having previously written upon them, in November, 1928, he abstracted chronologically the literature relative to sand blasting, silicosis, dust problems and allied subjects, covering the 20 years previous to 1928. He also prepared and edited an enormous amount of material in the form of monographs and papers, as well as informal talks for various regional and local meetings, and a huge amount of popular health material in the form of short health talks, given at various meetings, syndicated for industrial plant publications, sent to newspapers, and prepared for special occasions or specific uses.

Committees were also an important phase—both inside and outside the Council offices. One, which took a great deal of time and effort, was the Sand Blasting Committee, through which research was done on methods of sand blasting. As one result of this direct contact with the technical and popular aspects of industrial health and public health activities, a considerable number of files of standard procedures were evolved for advice and help to various groups. Among these was one on "Health Courses and Campaigns in Industry."

An interesting development of the work of the National Safety Council was the visits to manufacturing plants. More than 60 of such visits were made by Sappington throughout the United States during a period of a few years, and many kinds of industries were studied first hand.[3] This gave him an otherwise impossible breadth of vision regarding manufacturing processes, and was an extremely valuable experience for a consultant who is both physician and industrial hygienist—since plant processes are of vital importance in the study of any health hazard in industry.

During this time also, affiliations exclusive of the regular professional and medical contacts were kept up with many important groups and associations.[4]

Educational activities included lectures given by invitation at various universities, among them the University of Illinois Medical School, in Chicago, and the School of Public Health, University of Michigan, Ann Arbor; and the preparation of material for teaching in various courses at Harvard University, Boston, Queen's College, Toronto, Canada, and the University of Iowa.

International contacts were maintained. Representatives of various schools of public health called for consultation and exchange of ideas, representing almost every foreign country. Some of these were sent by the Harvard School of Public Health after having finished courses and before going back to their native lands to assume high positions in this type of work. Others came voluntarily and directly from schools or from various industries in foreign countries.

In addition to making endless and innumerable contacts with industrial groups and organizations, Sappington early realized that the casualty insurance companies were to play a very prominent part in the insurance and compensation angles of industrial medicine. This led to many conferences, interviews, and visits, as well as voluminous cor-

3. These included: cameras and accessories; meat packing; automotive; commercial-stores; food products; abrasives; electrical devices; telephone instruments; rubber; leather; toiletries; boilers; oil; aluminum; sugar; airplanes; safety equipment; stationery; foundries; tobacco; glass; cooking utensils; steel; cement; silk; lead; zinc; butchers' supplies; railroads; pipe and tubing; fiber products; chemicals; paper; water power; storage batteries; cash registers; refrigerators; pumps; milling machinery; axles; chain belts; sanitary ware; telephone companies; breweries; Pullman car manufacturing; telephone and engineering laboratories; rayon.

4. Among these were American Management Association; Illinois State Society for Mental Hygiene; National Association of Commercial Organization Secretaries; American Red Cross; Michigan Association of Industrial Physicians and Surgeons; New York State Society of Industrial Medicine and Surgery; Chicago Board of Health; American College of Surgeons; Chicago Tuberculosis Institute; Philadelphia Health Council and Tuberculosis Association; American Association of Railway Surgeons; American Chemical Society; National Association for Public Health Nursing; Mellon Institute; U. S. Bureau of Mines; U. S. Public Health Service; Industrial Relations Association of Chicago; Illinois State Industrial Commission; American Dental Association; Chicago Nurses Club; Cleanliness Institute; National Electric Light Association; Bureau of Tuberculosis, California State Department of Health; Bureau of Safety; Union Health Center, New York City; Rockefeller Institute; Bureau of Industrial Hygiene, Mcihigan State Department of Health; Los Angeles Health Department; Chicago Society of Industrial Medicine and Surgery; Stanford University; Management and Engineering Corporation; American Social Hygiene Association; Health Department, Baltimore, Maryland; Illinois Manufacturers Association; Life Extension Institute; American Gas Association; Mohair Institute; Wisconsin Manufacturers Association; Illinois Association of Commerce; New York Academy of Medicine; American Foundrymen's Association; U. S. Bureau of Standards; Special Libraries Association; Illinois Academy of Science; Bureau of Medical Economics and Council on Physical Therapy, American Medical Association; London School of Tropical Medicine and Hygiene; Portland Cement Association.

respondence, with leading insurance company representatives, both in Chicago and at their offices in various cities. The result is that there is probably no important casualty and indemnity insurance group in the United States that has not at some time been exposed to his ideas.

MEANWHILE evidences of national and international recognition were becoming apparent. At the age of 40 his biographical sketch went into "Who's Who in America," followed the next year by a similar sketch in "Who's Who in Chicago." In 1933 he was included in "American Men of Science," a biographical directory issued by the American Association for the Advancement of Science, in which he was elected to fellowship in 1933. There was also a biographical sketch in "Illinois—The Heart of the Nation," published in 1933. He was a delegate to the International Hygiene Congresses at Dresden in 1930; a member of the VI International Congress on Accidents and Industrial Diseases at Geneva in 1931; and an associate member of the VII International Congress on Accidents and Industrial Diseases at Brussells in 1935.

Among activities, he was also Chairman of the Committee on Ten-Year Research Program of Nursing Service in Industry, sponsored by the National Organization for Public Health Nursing in New York City; and Sectional Councilor of the Industrial Hygiene Section of the American Public Health Association. He has been a member of the Board of Editors of *The Sight Saving Review* since its inception; and a member of the Resolutions Committee of the Industrial Hygiene Section of the American Public Health Association, and of the Committee on Industrial Poisons of the American Association of Industrial Physicians and Surgeons, besides doing committee work on other special boards.

In his professional affiliations, he is a Fellow of the American Medical Association, the American Public Health Association, and the American Association for the Advancement of Science; a member of the Chicago Medical Society, the Illinois State Medical Society, the American Association of Industrial Physicians and Surgeons, and the Illinois Academy of Science. He is a charter member of Delta Omega, public health honorary society, and Phi Gamma Mu, an honor society in the social sciences.

SAPPINGTON has been a prolific writer, but not at the expense of unity and concentration on the given subject; there has been no dilution of thought, no scattering of perspective, but a continual training of the gunsights on a definite bull's eye. With always a very good reason for writing any article or paper, the material was well organized and thought through to definite conclusions before writing was begun. He has written more than 200 separate and distinct papers and articles[5] on the many phases of industrial health, medicine and hygiene, and literally hundreds more of abstracts, editorials, current comments, questions and answers, book reviews, radio talks, papers, and almost every conceivable type of transmission of the written and spoken word in the field of the *industrial* application of hygiene, medicine and health, as well as occupational diseases. Although his work, and his writings, are mentioned in a number of books, and with increasing frequency in the more important books, Sappington has, himself, never written a book—not yet.

His philosophy in, and his outlook upon, industrial medicine and hygiene, past, present and future, can be gathered from his writings. They follow through—in any sequence of selections.

More than seven years ago he wrote: "Progress in organization is much behind that made in scientific development in modern scientific medicine. Thus coordinating agencies are sorely needed; and there is no more important problem in modern medicine than this particular deficiency. There is an especial need for official agencies to develop the idea of preventive medicine in industry to its fullest extent. This principle seemingly is not feasible in private medical practice because physicians have apparently not been successful in developing a demand for preventive methods in private work. Official agencies can take the initiative because they are not in the position of a physician endeavoring to increase his personal following. Perhaps the great weakness in private practice is not realizing that prevention is more important than cure. Industrial workmen are constantly searching for a guarantee against the occurrence of three unfortunate circumstances—sickness, unemployment and death. Perhaps sickness is the greatest of all these problems; certainly it may cause the other two, it occurs in greater amount, it costs more, and its effects are more disabling. The private medical practitioner is in a very peculiar economic position—he exacts money from his patients during the period when there is no income. The proper types of service in industry can meet this situation. But the industries must have a common ground of reference and consultation."

Again: "When we ponder upon the future of industrial hygiene, a conservative viewpoint would give to it unlimited chances for the betterment of human kind. It stands at the very forefront of preventive medicine, in the very vanguard of public health effort; it has the possibility of correlating many other forms of health supervision; indeed it is the liaison officer in the regiment of health service recruits."

The proper assessment of factors in industrial hygiene and health work, especially the adequate interpretation and use of such factors, has become a very vital part of his work. He has not been able to forget the words of Dr. W. C. Rappleye[6]: "The quality, interpretation, and correlation of scientific knowledge depend upon trained personnel who know the significance of that knowledge and how to use it." His own statement of this principle is: "The control and prevention of occupational disease is of the greatest consequence, and probably the most important aspect of the whole subject of the significance of occupational diseases. Quite likely the progressiveness of any industry will be largely determined by the application of all possible means and methods in the control of diseases due to occupational influence. The specific means for such control and prevention lie in the intelligent correlation of our present available methods in the plant, laboratory and the clinic, and, as has been stated before, the integration of

5. About three dozen of the most important of these are named in Dr. Sappington's biography in WHO'S WHO IN INDUSTRIAL MEDICINE AND TRAUMATIC SURGERY, 1936.

6. In the *Bulletin of the American Medical Association,* October, 1930.

such information through the industrial physician. The socio-economic implications of occupational diseases in their various aspects make this portion of the problem assume the greatest importance of any factor with which we are concerned. In short, prevention and control are the most economical procedures viewed from any angle."

Again: "Concerning the apparent despair and discouragement in the minds of a number of persons who have had troubles either with silicosis or alleged silicosis, it is not stretching the truth to say it is just as presumptuous to believe that you can do nothing as it is to think that you can do everything. Surely there is a middle ground where one can profitably use the collective intelligence available and accomplish much more than by the presumptiveness of the extremes mentioned."[7]

And: "Since the Edison experience has shown that sickness causes 20 times as many cases of absenteeism as accidents and is responsible for seven times as much loss of time from work, it would seem rational that any program for solving the problem of lost time would give results, if money was intelligently used to further disease prevention. This does not mean that vigilance should be relaxed in the slightest degree in attempting to prevent accidents. Accident prevention has received so much attention in the past, however, that it now seems evident that emphasis might profitably be placed on earnest attempts to thwart the inroads of disease in industrial establishments."[8]

And again: "An approximately accurate measure of the severity of any given health hazard in industry has except in recent years been largely a by-product of presumption and conjecture—and in many instances this is still true. The reasons are several; there has been no real incentive for the development of clinical and laboratory knowledge regarding the effects of disease principally because the compensation regulations have been built up around the subjects of occurrence, prevention and treatment of injuries. Then, too, many physicians have not had at their disposal methods used in plant surveys and laboratory analyses for the measurement of the environmental exposure in the various industries. It is evident that these problems have demanded the 'bridging-over' of the two fields of medicine and engineering and the use of the sciences common to both, physics and chemistry. It is useless to expect the development of specialized personnel, in which individuals shall have the advantage of a thorough background both in medicine and engineering. It is not impossible, however, to develop specialists, who, having had a medical background, can acquire specialized information regarding occupational diseases and an appreciation and practical working knowledge of the laboratory methods required. Industrial medicine has for many years included within its scope such a point of view; no doubt, however, greater emphasis will be placed upon the use of continued improvements of these procedures in years to come, for not only do legal developments appear to sanction it, but economic preservation also demands it."[9]

Again: "The physician is and always will be the leading figure in the field of industrial health; as a professional technician he has gained recognition, but as an administrator it must be said that he has not clearly seen his responsibilities or his opportunities. Industry will not brook dictatorship, but medicine must officially present a united front if it would continue to assume responsibility and leadership in industrial medicine."[10]

And: "The administration of industrial medical programs shows all the permutations and combinations that are possible on a Chinese lottery ticket. Industrial relations groups claim that they are responsible for the administration of industrial medical and safety services; the safety departments feel that medical and health procedures belong under their jurisdiction; it is comparatively rare, but has occurred, that a physician has undertaken the management of all three activities. Probably the ideal arrangement is to place all three departments on an equal authoritative basis and make them all directly responsible to the general manager. I can see no large measure of success for any unless all three activities are constructively coordinated."[11]

And: "Industrial health supervision provides exceptional opportunities for the collection and analysis of morbidity material which is otherwise practically unobtainable. It is only through the careful measurement of this experience that we shall provide a rational basis for the inauguration of changes which must come if progress is to be made in industrial hygiene. Up to the present time, attention has been concentrated mainly upon accident experience. More thought and effort should be put upon the collection and analysis of material based on minor illness and physical defects. This data could form the background of rationalized preventive medicine in industry."[12]

And: "Industry is now assuming and will contine to more widely assume the responsibility for sickness in its workers. As one industrial hygienist has put it, the industrial physician 'ought to be projected into the great scheme of production and not forever played as a life saver.' There is no logical reason why the financially more important problems incident to sickness should not be made a part of the cost of production as has been the case with accidents; but most important of all is the service that provides for careful examination before employment, correction of physical defects, early treatment of minor ailments, and other procedures calculated to preserve the health and efficiency of the workers, upon whom the quality and quantity of production depend."[13]

And: "A recent survey of the schools in San Francisco . . . . has shown that 92% of the pupils leave the public school and directly enter industrial work . . . . Hayhurst has quite recently suggested that perhaps there is a lack of adequate health supervision for children between 12 and 16 to 20 years of age—this being the period of greatest importance in the correction of serious defects and chronic afflictions. If different groups of children could be examined while in school and the records preserved and compared with ex-

7. "Order from Chaos—The Use of Scientific Methods in the Prevention, Control and Defense of Silicosis Cases," *Industrial Medicine,* March, 1934.

8. See Footnote 1, on p. .....

9. "The Control of Occupational Diseases by Laboratory Methods." *Journal of Industrial Hygiene,* January, 1935.

10. "The Industrial Physician and the General Practitioner." *Journal of the American Medical Association,* December 27, 1930.

11. "A Diagnostic Study of Industrial Medicine," *California & Western Medicine,* December, 1932.

12. "Industrial Morbidity Data and the Physician," *American Journal of Public Health,* July, 1927.

13. "Sickness versus Accidents in Industry," *California & Western Medicine,* April, 1928.

aminations made later in an industrial establishment, it would appear that we would have a fair estimate of the efficiency of health work in schools . . . . It seems rational to believe that the basis for industrial health work may, therefore, be largely grounded in school health supervision and that the human product may be improved in the direct ratio in which school health work becomes more efficient. Gradually this would cause industry to become more than casually interested in the raising of standards of school health; in fact, one may say that there might come a day when the larger industries would contribute in a material way to the advancement of health work in the schools. A real program of preventive medicine in industry could thus be developed."[14]

And: "The biological, chemical, physical and socio-economic phenomena of occupational diseases have never yet been critically analyzed nor carefully integrated on any extensive basis. If this statement seems to be a mere collection of words, even superficial consideration of it will reveal its hidden and startling implications. If any branch of medicine ever needed a modern Osler, it is industrial medicine today. . . . It can be readily foreseen that the development of a large body of data integrating the causal relationships with the physiological effects will ultimately become the basis for the determination of the principles of underwriting and compensation. Insurance companies which are going to handle the underwriting of occupational diseases—and they surely will be underwritten by certain groups—have come to realize this, and will be very active in the development of such data. It, therefore, behooves industrial physicians and surgeons to appreciate the etiological factors and to correlate these with the clinical records."[15]

THIS is the background, and the philosophy, of a specialist. And Sappington has emerged from it as a specialist. The background and the philosophy are somewhat lengthily while still selectively set forth because this man's career—planned in advance, and intensive and purposeful at every stage of its development—is a forceful demonstration:

First, of the fact that industrial medicine is not, in and of itself, a specialty in the practice of medicine, but is a field of practice in which there may be a specialist;

Second, how a man becomes a specialist in industrial medicine and its concomitants; and

Third, what such a specialist is.

In 1932 Sappington decided to open his own office for consultation work in industrial medicine and hygiene.

As a specialist he is ONE Simon-pure consultant. He has no private practice of medicine; he does not diagnose, prescribe or treat the individual patient. His work is altogether with the employment relation; all of it comes from that relation; all of it is confined to that relation.

And his work from that relation has three principal divisions. First is the study of the exposure to dusts, fumes, gases, vapors, etc.—in which these are measured and rated. Second comes the clinical side—physical examinations including chest x-rays and the necessary clinical laboratory tests to determine what effect the various types of exposures have on employees. And third is the coordination or correlation of the information thus obtained—in terms of cause and effect relationships for the purpose of prevention, and in connection with insurance underwriting, proposed legislation, and medicolegal problems.

This is what he does, and it is all he does. The study of the exposure, whatever it may be, as the cause; the manifestation of what takes place in the human body as a result of the exposure, as the effect; and the putting of the two together, in terms of prevention. And in the latter connection he has with reference to occupational diseases in general, the same forward view that he had toward his own career in particular. He realizes that it will be a long time before the field of industrial medicine is in possession of such a body of cause-and-effect data on occupational diseases as will permit insurance underwriting, for example, to be done on the same basis with reference to occupational diseases as has been the case for a number of years with reference to occupational injuries. But meanwhile, and pending standardization, as it were, in this respect, there is already in existence and available for use a sufficient amount of such information and data to serve as a foundation for the conclusion that the method of attack upon the problems above outlined is the rational method.

In Dr. Sappington's relations with INDUSTRIAL MEDICINE, he is its Consultant; and, more than that, he possesses, in a remarkable and unusual degree, the ability to participate in an important medical meeting and thereafter indite a masterly summary—concise and complete—of all that was read, said, and presented. Probably the fact that he is, himself, an experienced and accomplished speaker has something to do with his proficiency in this respect.

SAPPINGTON, the man, transcends biographical sketches; he exceeds his descriptions; he is bigger than estimates of him. This is commonplace among those who know him well, for he grows on acquaintance. At once alert and reserved, he has a nicely balanced blend of the judicial and the dynamic in his temperamental make-up. He is careful and far-sighted in his reasoning. He thinks quickly, but his conclusions are exact and precise, and compounded of the many elements of preparation and scientific habits of mind which have become second nature to him; he thinks things *through*. And the conclusions he arrives at are weighted invariably with his thorough possession of that inestimable but too rarely encountered quality of character, a high and conscientious sense of responsibility. An essential fineness is evidenced by his life-long devotion to music as a complete and fulfilling avocation; he is musician and composer.

And so this man is a specialist. Moreover he is a specialist with the courage to be just that—which is no small factor of the recognition that is now his in and from all of his contacts. He studied and got ready, and now he is busy—the surge of occupational disease interest and occupational disease legislation rolling in from all sides found *him* prepared. But his qualities are such as to make him a real illustration of the traditional belief: "If you want something done, give it to a busy man."

14. "Relation between Health Supervision in Schools and Industries," *American Journal of Public Health*, June, 1926.
15. "The Significance of Occupational Diseases," *Industrial Medicine*, June, 1935.

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Industrial Medicine

ALMOST trite is the comment that: "Occupational diseases are now where compensation for injuries was 25 years ago." This is true, but with the vast difference that the medical profession is, and for several years has been, aware of the importance of occupational diseases. There is, of course, some opinion that it required the recent silicosis hysteria to bring this about. But regardless of how it came, the interest of the medical men in occupational diseases is present, and wide spread, and intense. And it is being manifested in many ways, the most important being the increasing number of expressions regarding it on the part of organized medicine.

Not so very long ago the organization attitude in general was: "We ought to do something about it." Now, however, the picture is rapidly becoming one of things done and being done. Indeed it is rare, nowadays, for an important organization to omit from the program of its principal meetings the definite indications of a real interest in industrial medicine.

In this connection the work of the American College of Surgeons has a high place in the record of accomplishments. A brief account of its plan of operation, and the basis of its effectiveness, is included in this issue, from the "Approval Number" of the A.C.S. Bulletin. Interesting are its remarks: "In comparison to the safety movement in this country, industrial medicine has heretofore had a slumberous career . . . . In the last few years distinct progress has been made in industrial medicine . . . . An increasing number of employers and insurance carriers are realizing that *the best* medical and surgical service is the cheapest . . . . and *other* competent physicians and surgeons are being attracted to this type of work. In short, the care of the human being in industry is being transferred from the obsolete repair department to the modern maintenance department."

The italics in the foregoing are ours. We like to emphasize the connotations of "the best," and "other." Wide acquaintance among the men who have been in industrial medical work for some time is very persuasive toward the conclusion that industry in general and industrial employees in many particular instances are getting "the best" from the standpoint of talent in all branches of medical practice. On the mass basis of the patients' problems, as they are represented in any substantial employment situation, the employer can and does command "the best." And the fact that "the best" is ready at hand, prepared to be availed of, is proof that the profession itself has not been "slumberous" in the matter of occupational diseases. Recognition of this is contained in "other."

Industrial medicine, as a field of practice, has its experts, its specialists. It is no more the little sidewalk, indicated in the biographical sketch of one of its specialists elsewhere in this issue, as paralleling the road of preventive medicine traveled by the Public Health Service. On its own account it has broadened out into a wide and solidly founded highway, leading straight to the level plateau of economic satisfaction, and there proceeding through the garden of practical and economic humanitarianism.

"In short," industrial medicine has arrived: The field of medical practice in which all that medical science has heretofore been able to summon to its aid in treating the individual patient as a human being is now being brought to bear upon the patient as a human being *plus* the fact that he is a human being in economic dependence upon a contract of employment in which he is one of the contracting parties. The field not only of diagnosis, treatment and correction, but also the field of prevention *ab initio*, and thereafter of "modern maintenance" of freedom from the things prevented. The trend, as Dr. Selby so clearly points out, is toward the intensification of these objectives of this expression of practice.

We sometimes think along these lines to the almost political conclusion that the economic ideal would be an expansion of the present facilities of industry to the point where there would be no unemployment, because if this were to be brought about there would be little, if any complaint, regarding the lack of medical care on the part of any substantial portion of the population. "In short," again, the answer to the alleged lack in this respect seems almost to be: "Go to work! Get a job."

For along with the job goes not only medical care, but also the very best medical care that it is possible to obtain—and, as Dr. Selby shows, to the benefit, and not to the detriment of the medical man who does not encounter the patient in, of and from the employment situation.

Industrial Medicine and Surgery

Survey of Compensation Laws

SHARP conflicts among the laws of the various states governing compensation for workmen injured in industrial accidents, compensation for workmen contracting occupational diseases, and physical examination requirements for employment, are disclosed in the findings of a nation-wide legal survey made by the Air Hygiene Foundation. * This year-long study notes a liberal trend in some states to award compensation to sufferers of occupational diseases.

The survey, the most comprehensive of its kind ever undertaken, involved the examination of all legal literature for "statutes and court decisions respecting occupational diseases from air pollution." It was directed by Attorney Henry A. Middleton of a Toledo, Ohio, law firm (Williams, Eversman and Morgan) and supesvised by the Foundation's legal committee. The Foundation's headquarters are at Mellon Institute, Pittsburgh.

"The guiding principal in the early acts," the review continues, "was the awarding of compensation for accidental injury, meaning thereby traumatic injury. . . . In those years the importance of disability due to occupational disease was not greatly recognized.

"It however became increasingly apparent that 'injury' might be suffered gradually, and that the result of such general breakdown of the strength and resistance of the employee was more serious in many instances than traumatic injury.

Courts, therefore, in some states, wrestled with the problem of construing 'injury' so as to include disease, and bring disease within the compensation acts."

Maryland is cited as one of these states. There, it appears from court decisions, a diseased condition contracted in employment is compensable if caused by the negligence of the employer.

In discussing the confusion existing because of the differences in the laws, the report expains:

"Each state is a unit, a sovereign, and it handles its problems in its own way. Uniformity is lacking, although of course the statutes of many states are similar. The courts of the various states follow such precedents as appeal to them; consequently, on many important questions, there is likely to be a divergence of opinion among the courts, and often the courts fall into two or more groups, each group following a different theory with respect to the same legal problem."

The survey shows that Workmen's Compensation Acts have been passed in 46 states (Arkansas and Mississippi are without this type of legislation), while only 17 states and the District of Columbia grant compensation for occupational diseases. In five of the latter, however, silicosis is not included in the list of compensable diseases.

* From H. B. MELLER, Managing Director, Air Hygiene Foundation of America, Inc., Pittsburgh, Penna.

There is a legal obligation on the part of the employer to provide a safe place to work and usually the matter of ventilation is covered. The mining laws approach uniformity as to air quantity and mechanical ventilating equipment.

Concerning the controversial point of required medical examinations for workmen, the report comments:

"With the adoption of occupational disease acts, new medical problems arise and more adequate provision is required for medical examination and treatment. This is especially true with respect to dust disease, which calls for diagnosis by skilled specialists experienced in that field."

In New Mexico the compensation act says, "it shall be the duty of the workmen" to submit to physical examination, while in New Work, the Act of 1936 covering silicosis and other dust diseases, states:

"It is hereby declared to be the policy of the legislature of this state to prohibit, through every lawful means available, any requirement as a prerequisite to employment which compels an applicant for employment in any occupation coming within the purview of this article to undergo a medical examination."

Missouri provides for examination as often as once a month, by a competent, licensed and reputable physician, of all employees who come into direct contact with certain poisonous agencies or injurious processes. Three states, New Jersey, Ohio, and Pennsylvania, require employers to have examined, at least once a month, employees engaged in any work or process exposing them to lead dust, lead fumes or lead solutions.

North Carolina requires examination both prior to employment and from time to time during employment.

In five states, Kentucky, Massachusetts, New York, North Carolina, and West Virginia, definite recognition has been given to some type of medical board composed of specially qualified physicians and surgeons, and charged with the duty of diagnosing occupational disease and of advising the administrative board or commission with respect thereto.

The right of a workman who has suffered an occupational disease due to the negligence of the employer, to recover damages at common law, is another point on which the courts hold contrasting views. In many states there appears to have been no occasion for the courts to rule on the subject. In other states where the right existed, it has been modified by the passage of workmen's compensation octs. In three states, Illinois, Michigan, and Ohio, it has been held that such common law right to recover damages for occupational disease never did exist. North Dakota may also be included here.

After emphasizing that "lawyers might well differ as to where any particular states belong," the survey adds:

"In three states, Connecticut, Georgia, and Texas, we would say that recovery is extremely doubtful.

"The common law right to recover for occupational disease caused by negligence of the employer probably exists, although there is some doubt, in the following 12 states:

"Delaware, Iowa, Maine, Maryland, Massachusetts, Oklahoma, Oregon, Pennsylvania, Rhode Island, Tennessee, Virginia, and Washington. That such right does exist seems well settled with respect to another group of 12 states: California, Kansas, Kentucky, Louisiana, Minnesota, Mississippi, Missouri, Nebraska, New Hampshire, New Jersey, New York, and West Virginia.

"It should be note, however, that occupational disease is compensable to some extent, under the compensation acts, in all of this group except four states: Kansas, Louisiana, Mississippi, and New Hampshire. Hence the common law right to recover in eight of the 12 states is limited."

In the remaining 17 states and the District of Columbia no court decisions deciding the question of common law right could be found.

Summary

Each state is a unit—a sovereign—and it handles the problems in its own way. Uniformity is lacking, although of course the statutes of many states are similar. The courts of the various states follow such precedents as appeal to them; consequently, on any important question, there is likely to be a divergence of opinion among the courts, and often the state courts fall into two or more groups, each group following a different theory with respect to the same legal problem. All of this demonstrates the necessity of studying the statutes and court decisions of each state to determine the status of the law in that state and also the impossibility of accurately tabulating and classifying the laws with repect to dust hazards.

A few general observations however may be made.

Workmen's Compensation Acts have been passed in 46 states. The Longshoremen's and Harbor Workers' Compensation Law enacted by Congress in 1927 and the Workmen's Compensation Law of the District of Columbia enacted in 1928 provided compensation with respect to employments within the District of Columbia and also with respect to disability or death resulting from injury occurring upon navigable waters of the United States, where such compensation is not provided by the states laws. Those two acts, being identical as to coverage, are commonly referred to as the Workmen's Compensation Law of the District of Columbia.

There are no Workmen's Compensation Laws in Arkansas and Mississippi. The first states to pass a Workmen's Campensation Law was Wisconsin. The law became effective May 3, 1911. The last state was South Carolina, whose act became effective September 1, 1935. The State of Wisconsin pioneered to a large extent; and occupational disease has been compensable in that state, by statutory enactment, since 1919.

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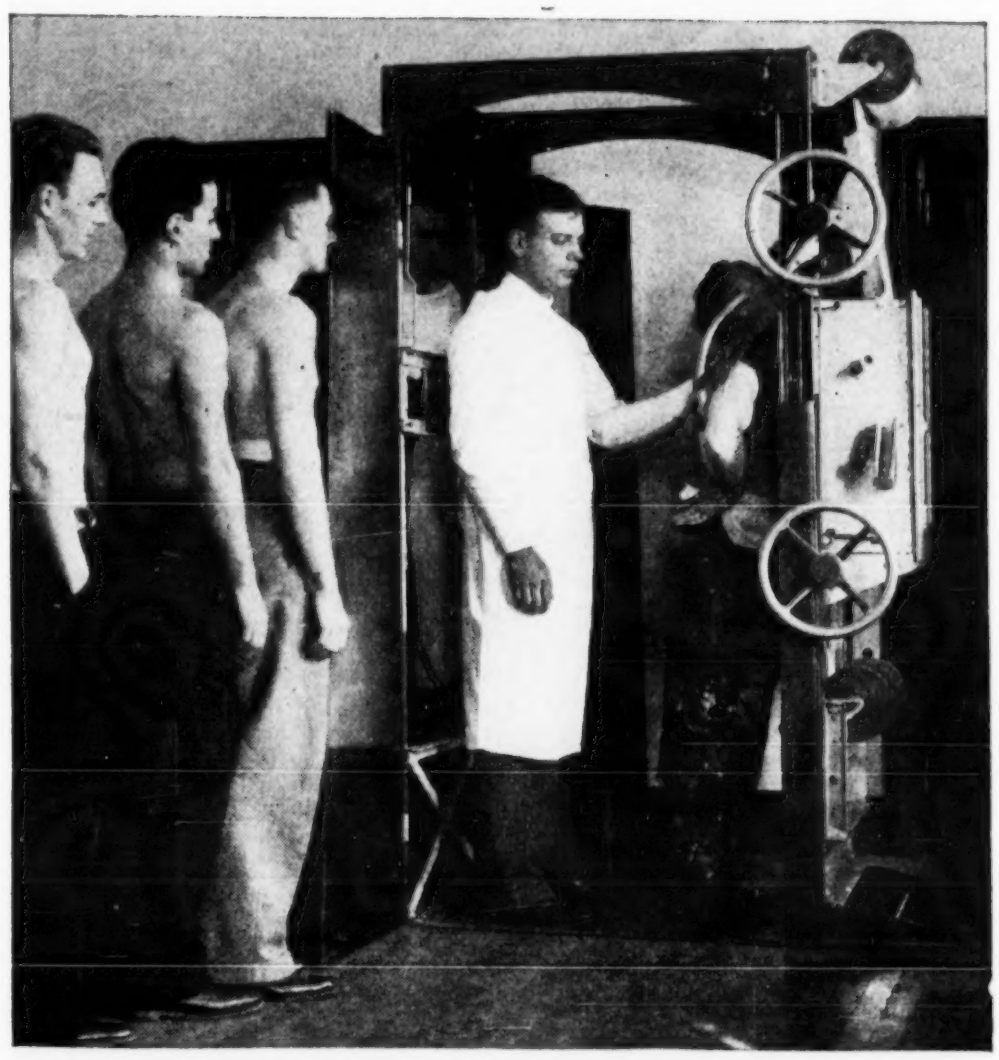

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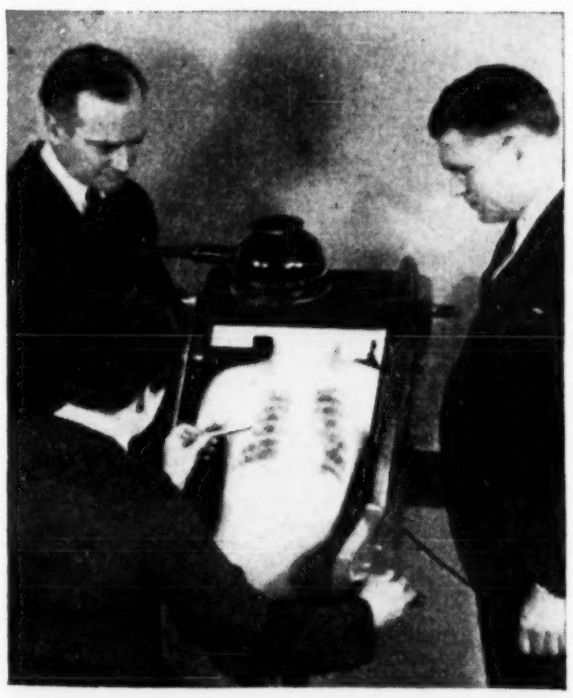

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The compensation acts are compulsory as to private employers in 15 states and they are elective as to private employers in 31 states. Insurance to cover the risk is compulsory in seven states. A monopolistic state fund, out of which compensation is paid, is maintained in seven states, towit: Nevada, North Dakota, Ohio, Oregon, Washington, West Virginia, and Wyoming; but, in two of those states (Ohio and West Virginia), the employer may, by proving financial responsibility, carry his own risk with limited contribution to the state fund for the expense of administration. In 11 states, a state fund is maintained, but the employer is not required to avail himself of its privileges and may provide compensation otherwise. In 28 states, there is no provision for a state compensation fund. The administration of the compensation acts is vested in a commission, board, or single commissioner in 40 states. In six states, the acts are administered by the local courts.

The guiding principle in the early acts was the awarding of compensation for accidental injury, meaning thereby traumatic injury. In those years, the importance of disability due to occupational disease was not generally recognized. It however became increasingly apparent that "injury" might be suffered gradually, and that the result of such gradual breaking down of the strength and resistance of the employee was more serious in many instances than traumatic injury. Courts therefore, in some states, wrestled with the problem of construing "injury" so as to include disease and bring disease within the compensation acts. An outstanding example of such court effort appears in the Maryland decision rendered in 1925 in the case of Victory Sparkler & Specialty Co. v. Francks. The court held in that case that phosphorus poisoning resulting from exposure, over a period of years, came within the term "accidental injury" because the injury was suffered by chance, that is, not foreseen or anticipated by the injured person, and found that the condition resulted from the negligence of the employer. By this reasoning, the court held that the particular disability was compensable, although it held it not to be an occupational disease. It was merely an "accidental injury." The result seems to be that, in Maryland, a diseased condition is compensable if caused by the negligence of the employer.

Leaving Maryland in a class by itself, it is found that occupational disease is compensable, to some extent, in 16 states and the District of Columbia. They are: California, Connecticut, Illinois, Kentucky, Massachusetts, Minnesota, Missouri, Nebraska, New Jersey, New York, North Carolina, North Dakota, Ohio, Rhode Island, West Virginia, and Wisconsin. In five of this group, to-wit: Minnesota, Nebraska, New Jersey, Ohio, and Rhode Island, silicosis is not included among the compensable diseases. In West Virginia, silicosis is the only occupational disease made compensable.

We have discussed, at some length, the matter of physical examination and functioning of medical boards in connection with occupational disease. As pointed out, only five states, to-wit: Kentucky, Massachusetts, New York, North Carolina, and West Virginia, have created what may fairly be called "medical boards", while New Jersey, Ohio, and Rhode Island provide for better than the ordinary type of medical examination, but do not have provisions for medical boards.

The labor laws and factory inspection laws of the various states recognize the legal obligation to provide a safe place to work, and ordinarily the matter of ventilation is featured. Failure to comply with these laws results in liability of the employer, and frequently authorizes the assesment of penalties. The mining laws approach uniformity as to the quantity of fresh air required and as to the mechanical equipment required in connection with ventilation. An effort has been made in this survey to cite the mining laws so that they can readily be found and to give the substance of them, but in most instances, it has been impossible to quote the laws verbatim.

Administrative rulings and safety rules have been adopted by numerous administrative bodies, and, in each state, an effort has been made to quote the substance of such rules or to state what administrative body made the rulings and how they can be procured where they are too voluminous to quote or digest.

The common law liability for occupational disease is at once important and difficult to state concisely. In many states, there appears to have been no occasion for the courts to decide whether or not the right exists, at common law, to recover for occupational disease due to the negligence of the employer. In other states, where the right existed, it has been materially modified by the passage of Workmen's Compensation Acts. In three states, it has been held that such common law right to recover for occupational disease never did exist. At best, nothing more than a rough classification can be made of the states of the Union with respect to the common law right to recover for occupational disease. We are making such rough classification, but with the emphatic caution that the law of each state must be carefully studied, and that lawyers might well differ as to where any particular state belongs in such classification.

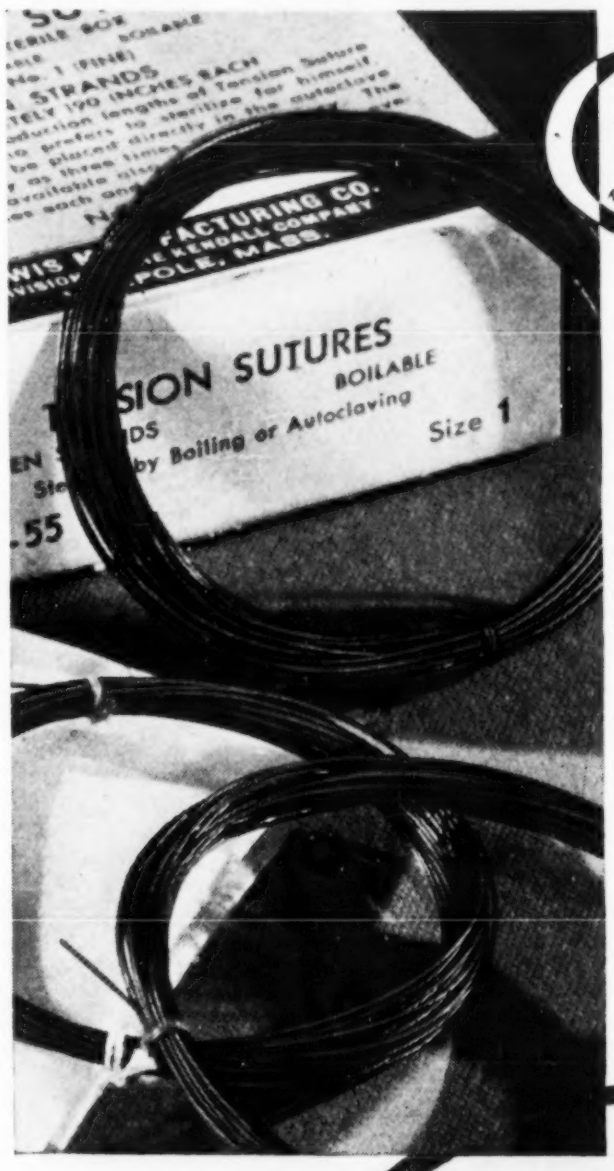
First, the states in which the courts have held that no such common law right of recovery exists: They are: Illinois, Michigan, and Ohio. North Dakota also probably walls within this group, not because the common law right never existed there, but because, if it ever existed, it has been completely superseded by the Workmen's Compensation Act. In that respect, the law of North Dakota is rather unique. In many instances, the compensation acts provide that the remedies given thereunder are exclusive; but they also provide that, if the employer does not comply with the act, the employee may pursue such remedy as he has at common law and the employer is, in such action, denied the common law defenses of assumed risk, fellow servant rule, and contributory negligence. Under such circumstances, it manifestly remains important to know whether any common law right of recovery exists in favor of the employee against such employer who has not complied with the act. In North Dakota however, the courts have held that the Workmen's Compensation Act is mandatory and exclusive; that an employer failing to comply with the act does not thereby become liable to a common law action but is liable to an action for compensation, as provided in the act, to which action lack of negligence is not a defense and which action is the sole and exclusive remedy of the employee. It may be said therefore that there is no common law right of action for occupational disease in North Dakota. The employee has only such rights as are given by the Workmen's Compensation Act. Occupational disease is compensable under that Act.

In three states, we would say that the common law right of recovery is extremely doubtful. They are: Connecticut, Georgia, and Texas. The situation in Georgia is also interesting as demonstrating the significance of court interpretationas bearing upon the common law right of action. The courts of that states have held that the remedy given under the Workmen's Compensation Act is exclusive, both with respect to disabilities for which compensation is provided and with respect to disabilities incurred in the course of employment even though compensation therefor is not provided. In most states, the courts have held that the right to recover at common law, if that right ever existed, is not barred by the provisions of the compensation acts, except as to thoe disabilities for which compensation is provided. In Georgia, occupational disease is not compensable under the Act. Nevertheless, any common law right to recover therefor, which may have existed, is apparently wiped out by court construction that the remedies given under the act are exclusive.

The common law right to recover for occupational disease caused by the negligence of the employer probably exists, although there is some doubt, in the following 12 states: Deleware, Iowa, Maine, Maryland, Massachusetts, Oklahoma, Oregon, Pennsylvania, Rhode Island, Tennessee, Virginia, and Washington.

That such right does exist, seems well settled with respect to another group of 12 states, to-wit: California, Kansas, Kentuck, Louisiana, Minnesota, Mississippi, Missouri, Nebraska, New Hampshire, New Jersey, New York, and West Virginia. It should be noted however that occupational disease is compensable, to some extent, under compensation acts, in all of this group, except four states, to-wit: Kansas, Louisiana, Mississippi, and New Hampshire. Hence, the common law right to recover in eight of the 12 states is very much limited.

In the remaining 17 states and the District of Columbia, to-wit: Alabama, Arizona, Arkansas, Colorado, Florida, Idaho, Indiana, Montana, Nevada, New Mexico, North Carolina, South Carolina, South Dakota, Utah, Vermont, Wisconsin, and Wyoming, no court decisions which decide the question as to whether or not such common law right exists can be found. It should again be



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noted that occupational disease is compensable in three of this group, to-wit, District of Columbia, North Carolina, and Wisconsin.

In conclusion, we may say that the making of this survey has been a pleasant, though difficult, task and that the manifest effort on the part of ligislatures, commissions, and courts to solve the rather recent disease problems arising out of industry should be a matter of great satisfaction to employers and employees alike.

Dermatitis Venenata and Compensation

THE extremely high incidence of dermatoses arising from occupation has come very prominently to the fore in this state in the last few years since compensation has been granted for disability arising from these conditions.* There are many types of skin diseases which might be included in this category—as for example, anthrax arising from the handling of infected skins or wool; acne arising from contact with such substances as mineral oil, paraffin or halowax, and so forth. By far the most common skin disease met with in industry, however, is that known to the medical profession as dermatitis venenata. This closely resembles poison ivy, and is due to exposure of the skin to an external irritant, usually a chemical to which the person is unduly sensitive or allergic.

It is frequently a difficult matter to determine which of the many substances with which a worker comes in contact at home and in his work is responsible for the skin condition which he develops. Where the question of compensation arises, it is not sufficient for the physician to merely cure the patient. He must be able to establish causal relation between his patient's dermatitis and one of the substances with which he comes in contact in his work—if such a relationship exists. If the patient's skin disease is the result of contact with irritants at home or elsewhere outside his workroom, he is obviously not entitled to compensation. The nature of the particular irritant which caused the trouble cannot ordinarily be determined by an examination of the skin lesions, since all cases of dermatitis venenata resemble one another very closely regardless of the causative agent. In an effort to devise a means for determining the nature of the irritant responsible for a given case of dermatitis venenata, the patch test has been developed and has proved of great value.

The present discussion is directed toward a brief review of some of the principles underlying the reactions of the skin to various known types of irritants as they occur in industry, and an appraisal of the patch test as a means of differential diagnosis of these conditions, with a consideration of limitations.

Skin Irritants Classified

ROSSER WHITE has classified skin irritants as follows:

1. Mechanical or physical agents which are irritating to the skin, such as heat, cold, friction, etc.
2. Keratin solvents such as akalis, oils, alcohol, aniline, aromatic hydrocarbons.
3. Dessicators and anhydrides such as potash, soda, glycerine and the like.
4. Subtances which are electrolytic or hydrolytic such as hydrochloric acid, cocaine, mustard.
5. Protein precipitants and irritants such as tannic acid, alkaloids and salts of the heavy metals.
6. Oxidizers which are poisonous to protoplasm.
7. Reducing agents.
8. Substances which form toxic nitro-derivatives such as TNT, picric acid, etc.
9. Neoplastic or keratogenic substances such as arsenic, petroleum, anilin, etc.
10. Biotic agents manifesting vegetable or animal activities on the skin. This includes micro-organisms and parasites.
11. Sensitizers. In this group fall the various allergic dermatoses known as dermatitis venenata.

R. L. Mayer of Breslau has listed the following substances among those capable of causing a sensitization dermatitis, of the eczematous type—in other words dermatitis venenata:

1. Solid insoluble substances as: textiles, leather, plants.
2. Artificial fertilizers.
3. Washing materials such as: patent powders, bleaches, etc.
4. Oils, shellacs, solvents.
5. Acids and alkalis.
6. Woods.
7. Flour bleaches.
8. Aromatic oils.
9. Alkaloids.
10. Drugs and medicants.

*MAY R. MAYERS, M.D., of the Division of Industrial Hygiene, State of New York Department of Labor; published in *The Industrial Bulletin*, May, 1936.

THE BULLETIN

of

The American Association of Industrial Physicians and Surgeons

THE object of this Association shall be to foster the study and discussion of the problems peculiar to the practice of industrial medicine and surgery; to develop methods adapted to the conservation of health among workers in the industries; to promote a more general understanding of the purposes and results of the medical care of employees, and to unite into one organization members of the medical profession specializing in industrial medicine and surgery for the mutual advancement in the practice of their profession.

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In investigating cases of dermatitis venenata—whether they occur in industry or outside—one is immediately impressed with the fact that there are apparently great differences in susceptibility among individuals to given irritants. We are all familiar, for example, with the fact that some people are susceptible to poison ivy when they go to the country while others never get it regardless of exposure. The same holds true for the other irritants responsible for dermatitis venenata. This hypersensitivity or hypersusceptibility of a given individual to substances which do not ordinarily injure normal people is known as "allergy."

Allergy is an acquired state in which there appears to be a physiochemical antagonism to specific agents, though the skin may be entirely normal to ordinary irritants. The reaction is a specific one to the particular substance to which the patient is allergic. The severity of the reaction depends not so much upon mass concentration (though this may also be a factor) but essentially upon the degree of hypersensitivity of the individual exposed. In a hypersensitive individual a very small amount of the substance is capable of producing disproportionately severe skin reactions.

Allergic Reactions

FOODS are equally responsible for allergic reactions. The so-called "strawberry rash" is well known to occur regularly in certain individuals whenever they eat strawberries. Eggs, milk, sea food and many other common articles of diet are not tolerated by everyone. Similarly, peculiar reactions due to personal idiosyncrasy, or individual hyper-susceptibility occur constantly in medical practice in relation to drug therapy. Such an old household remedy as castor-oil has been known to cause serious illness in susceptible individuals. Quinine and sodium salicylate are other examples of common drugs which may cause toxic symptoms in a few hypersensitive individuals, while large numbers of others take them in considerable quantity with great benefit.

Allergic phenomena may also manifest themselves in acute reactions in other parts of the body. When the respiratory tract is involved we have such well known diseases as hay fever or asthma. These diseases may develop as a result of hypersensitiveness to such substances as pollens from plants, horse hair, rabbits' hair, furs, feathers, etc. Certain individuals, for example, cannot enter a stable without an acute attack of hay fever. Others cannot with immunity sleep on feather pillows. Still others may not keep canary birds, rabbits, dogs, cats or other animal pets. Thus furriers, workers in the clothing industries who are exposed to wool dust, stable men, gardeners, florists or persons who work with animals may if susceptible suffer from hay fever or asthma.

From the standpoint of the various types of reaction of the skin to irritants, individuals may be classified in a general way into three main groups:

1. Individuals who are non-allergic and have normal skins. Such individuals will develop a dermatitis only if exposed to primary irritants in sufficient concentration—the severity of the reaction depending upon the mass dosage, the strength of the irritant, its concentration and the duration of exposure.

2. Non-allergic individuals having what is known as a sub-normal skin. Such individuals have either con-

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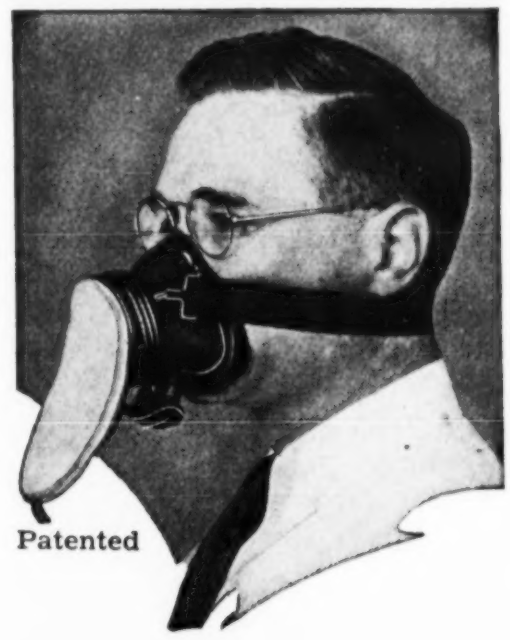

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genital or acquired structural defects or dysfunction of the skin. They are more easily affected than persons with a normal skin by ordinary common irritants, and tend to react more violently than normal when exposed.

3. Truly allergic individuals. In such persons the skin may appear quite normal and may react in a normal way to ordinary irritants. Once having become sensitized to a particular substance, however, a dermatitis venenata will appear when the individual comes into contact with this substance. A small exposure may result in a tremendous skin reaction in such persons. Furthermore, sensitivity while beginning with one substance may become polyvlent, i.e., it may tend to spread to other substances as time goes on.

In individuals who tend to be allergic the development of a dermatitis venenata will be facilitated under certain conditions. For example:

(a) The presence of a fungus infection (dermatophytosis, or athlete's foot) may be a precipitating cause of a particular eruption. It does this by interrupting the continuity of the skin; by reducing the natural resistance of the skin in a non-specific manner; and by setting in motion a sensitization mechanism which though first directed towards the fungus itself, later tends to become polyvalent so as to include numerous other substances as well.

(b) Trauma is a common factor known to precipitate an allergic reaction in an individual in this group. Experimentally allergic reactions have been precipitated by scarifying the epidermis. In industry one observes again and again the situation of a worker who has been in contact with a given substance for many years without ever having had any dermatitis. Suddenly, however, following a severe cut, burn, or abrasion a true dermatitis venenata appears. The worker suddenly acquires a hypersensitivity to a substance with which he has worked for many years without apparent ill-effects. Burns, abrasions, cuts, scratches, heat, moisture and cold are some of the factors which, by breaking the continuity of the skin, cause it to become more permeable to sensitizing agents. Workers whose hands are constantly immersed in water very commonly become hypersensitive in this way.

(c) Systemic causes predisposing to allergic reactions are not as well understood. However glandular disturbances resulting in excessive perspiration, or excessive dryness of the skin, seem to play a role in increasing the sensitivity of allergic individuals and so predisposing to the development of a dermatitis. Diabetes appears to make allergic individuals hypersensitive—particularly to the yeasts and to staphylococcus organisms.

(d) Primary irritants pave the way for sensitization and may also contribute to the production of a dermatitis. Under this heading are to be found strong caustics; alkalis and acids; rubefacients; mechanical irritants; radiant energy, such as ultraviolet light, radium or x-rays, and solutions which remove the grease from the skin causing it to become exceptionally dry.

It is of interest to note that cases

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of skin disease falling into the group under discussion—i.e., cases of dermatitis venenata—appear to follow definite laws of allergy. For example, it is well known that a rough incubation period seems to exist before the onset of a dermatitis venenata in individuals coming into constant intimate contact with the substance which later causes the trouble. It has been observed, for example, that many workers who develop a dermatitis have their first attack only after two or three months of contact. In some cases the dermatitis starts after a few days; in others, after a week or two. It appears to be only in the exceptional case that the eruption seems to break out immediately.

Another principle known to students of alergy is the phenomenon of flux insensitivity. This, too, has been observed by examination of workers in industry. Some of the workers, after their first attack never seem to have a recurrence. Most of the others, however, appear to be troubled only intermittently. In still other cases, the severity of the dermatitis in successive attacks increases; in others it decreases, and in still others there seems to be no change.

Bloch's work shows that susceptibility to primrose though directly determined by the make-up of the individual may also be largely influenced by the concentration and repetition of contact. He believes that, although some individuals may be more easily sensitized to a given substance than others, all can be sensitized if the concentration and the number of contacts is sufficient.

Case Histories

THIS principle was borne out to some extent in a recent series of examinations in a plant where workers were exposed to bakelite dust:

1. One man who had never had any skin disease previously developed a severe attack of dermatitis when the exhaust fan, over the drum where he had always been working, got out of order. The mass dosage of dust to which he was suddenly subjected was apparently too much for his resistance.

2. Another man put on a respirator which had been previously worn by some one else, and which was thoroughly saturated with dust. He got a dermatitis for the first time though he had been exposed to the same substance for years. The dermatitis was confined to his mouth and chin and soon disappeared. He had no subsequent attacks of dermatitis.

3. One worker got his only attack of dermatitis after he entered a dust-filled drum. The dermatitis soon cleared up. He has never entered the drum again and has never had a recurrence of his dermatitis though he has been in constant contact with the same substances.

4. There was general agreement among the men that when they were on twelve hour shifts they had many more cases of dermatitis than on the present eight hour shifts where total exposure was less.

Contributing Factors

IT HAS already been pointed out that scarification of the skin has been shown experimentally to sensitize it if a sensitizing substance is then rubbed into it. Recent examinations have shown that where a chemical substance has a gritty consistency, and the workers tend to get it pretty thoroughly rubbed into their skins in the course of their work, the incidence of dermatitis is unusually high.

The effect of heat and excessive sweating has always been known to be a contributing factor in the development of a dermatitis venenata. Recent experience in New York state has shown that the incidence of dermatitis venenata among workers tends to be definitely greater in the summer than in the winter. Furthermore, dermatitis tends to be more prevalent among workers tending machines which give off heat, than among workers in other departments.

The importance of an allergic history as a predisposing factor in the development of a dermatitis venenata is a question which has not yet been definitely settled. In a recent series of cases studied, however, there were several points of interest along these lines. One of the men examined, a chemist, who suffered from multiple sensitization through contact with numerous chemicals had a persistent case of dermatitis while working, which always cleared up on vacation. He gets an attack of asthma and conjunctivitis as well as an excerbation of his dermatitis with pruritus (itching) whenever he handles a chemical known as demethyl-ortho-cresol which is a resin base. The asthma and conjunctivitis are allergic manifestations also. Both

OCCUPATIONAL DISEASES

The General Assembly of the State of Illinois has recently passed an Act, covering occupational diseases of Industrial employees. This Act is particularly concerned with diseases of the lungs, and poisoning from certain chemicals.

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this man and another worker with a bad case of persistent dermatitis had a child suffering from eczema—also an allergic disease. Personal histories obtained of allied allergic conditions such as hay fever, asthma or eczema were too few to permit to follow this question further. Certainly on the basis of present knowledge an allergic history would suggest nothing definite as to an individual's susceptibility to a dermatitis venenata.

Patch Tests

PATCH tests were first devised by Judasson and are carried out as follows:

A small amount of the substance to be tested is held in place on the skin by a square of adhesive on the centre of which a slightly smaller square of some non-irritating, non-absorbing material such as oil silk or cellophane had been placed. These are left in place for 48 hours. They are then uncovered and a reading made. When possible, a second reading should be made about 20 minutes after the "patch" has been removed. When the reaction is negative it is further desirable to make additional readings every few days for a week or two if this can be arranged.

The adhesive sometimes causes a certain amount of irritation of the skin. A square of oil silk or cellophane is used therefore, to protect the skin immediately surrounding the gauze or linen on which the irritant under suspicion is applied so that if the test substance has caused a skin reaction, this reaction will be clearly differentiated from any reaction the adhesive may have caused.

Patch tests if they can be successfully used are potentially useful in two ways: (1) They might help in identifying allergic individuals and (2) by making it possible to discover the substances which are highly sensitizing they might assist in the substitution of other more innocuous materials. It has been found, for example, that the substitution of one type of soap for another — both equally effective for their purpose—will sometimes greatly reduce the incidence of dermatitis among the workers exposed.

The test is an easy one to apply but not so easy to read. In cases of great sensitivity when the substance causes a marked reaction, the reading is easy but in mild cases some experience is needed to rule out pseudo-reactions due to the adhesive; to recognize a true reaction accompanied by marked adhesive irritation, and to interpret very mild or negative reactions. The test has been found to be of value in eruptions of an eczematous type due to external irritants. It is also of value in some of the other eczematous eruptions, involving the upper layers of the skin in an inflammatory reaction even when the causative agent is not an external irritant. It is not, however, the method of choice in eruptions involving the cutis or middle layers of the skin, such as urticaria.

Valuable as is the patch test at the present time its limitations are very definite and must be well understood if it is to be used with any degree of accuracy in differential diagnosis. Limitations in the usefulness of this test may be summarized:

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1. One cannot be entirely sure that one has obtained all of the substances with which the individual has come into contact, for purposes of the test—particularly in an industry where large numbers of chemicals are used.

2. It is frequently difficult to reproduce the exact condition of work in terms of such factors as friction, scarification of the skin, temperature, humidity, perspiration, etc.

3. It is sometimes possible to get false positive reactions particularly in persons who at the moment happen to have a temporarily non-specific or generalized sensitivity, regardless of immediate exposure.

4. It is also possible to get false negatives as has been mentioned because the working conditions cannot always be duplicated; and also because individuals may be in what is known as a refractory or negative phase (non-hypersensitive phase) at the particular time that the patch test is made. Or the patch test may have been applied to a part of the body which does not seem to participate in the general hypersensitivity. Thus, where it is only possible to patch test an individual once, the results—particularly if negative—cannot be regarded as entirely conclusive.

These limitations in the usefulness of the patch tests are of tremendous practical importance. A single patch test which turns out to be positive is generally speaking a far better indication of sensitivity than one which is negative. In all cases where a patch test gives negative results, it is desirable that it be repeated after an interval, and on some other part of the body before definite conclusions are drawn as to lack of sensitization to the substance or substances being tested. This is not always practicable.

Clinical Test

WHAT is known as the clinical test, that is to say, the observation that every time a worker comes in contact with the substance in question he gets a dermatitis and that it clears up regularly when contact ceases is a far more definite diagnostic test. However, this, too, has its limitations because some workers, once having acquired a sensitization to a single substance may develop what is known as polyvalent sensitization involving other substances as well; or for various other reasons may have great difficulty in getting rid of the original dermatitis. In cases where both the clinical test and the patch test are negative it is desirable to wait until after the dermatitis is healed and then apply a patch test to the part which had been involved in the original dermatitis on the theory that sensitization at this point is to be expected if the individual is truly allergic.

In the various attempts made to rule out other skin conditions (not of occupational origin) for purposes of differential diagnosis — particularly in compensation cases—the question

as to the co-existence or pre-existence of a dermatophytosis or ringworm infection generally comes up for consideration. Ringworm may be diagnosed very accurately in the laboratory by examining scales removed from the lesions for the presence of the characteristic fungus which is the causative agent in this disease. When ringworm is found in workers claiming compensation for an occupational dermatitis it is frequently suggested that the case be disallowed as being non-occupational in origin.

Ringworm Infection

THE whole question of ringworm infection from this point of view will repay a brief examination. The recent extensive application of laboratory diagnosis to ringworm infection or dermatophytosis has demonstrated the fact that approximately one out of every five individuals has ringworm of the feet. Whether this high incidence represents a rapid increase in recent years—due perhaps to the widespread communal use of swimming pools, gymnasium lockers, showers and the like—or whether it merely represents a better appraisal of the situation because of the application of more exact diagnostic methods must remain an open question for the moment. That it is extremely common—and may be found by microscopic examination of scales removed from between the toes of persons entirely unaware that anything is wrong—has been definitely proved time and time again. In view of the widespread nature of this condition, therefore, it is entirely possible that a worker suffering from an occupational dermatitis, if examined also for the presence of ringworm, may very often be found to have both. The mere finding of the ringworm fungus cannot, therefore, be considered sufficient evidence for ruling out a possible coexistent occupational dermatitis.

Study of Relationship

THERE are, moreover, further aspects of the relationship of these two skin conditions which must be considered.

First, the mere presence of an occupational dermatitis is capable of so lowering the resistence of the skin as to predispose it to a secondary infection by the fungus of ringworm. In such cases, again, the fact that the fungus can be demonstrated very obviously cannot of itself rule out the possibility of a preceding and now coexistent occupational dermatitis.

Secondly, persons suffering from an initial ringworm infection or dermatophytosis will occasionally become hypersensitive to the fungus causing the infection. In such cases, for no apparent reason the dermatitis instead of remaining localized to the area where the fungus is, may suddenly spread over large areas of the body and cause disability. The fungus cannot usually be demonstrated in these areas. Such a condition is differentiated from "dermatophytosis" proper and is called "dermatophytid."

It is well established that "dermatophytid" may occur for no apparent reason in persons suffering from ringworm of the toes. It may also be initiated or precipitated in such persons by exposure to chemical irritants—even by exposure to sunlight. There may thus be no occupational basis for the generalized dermatitis in some cases. On the other hand the presence of the ringworm fungus apparently makes some individuals more sensitive than they would otherwise be to the chemicals to which they are exposed in the course of their occupation. It may well have been a particular occupational exposure in a particular case, therefore, which was the actual precipiating cause of the disability. Had the worker not been so exposed it is entirely conceivable that he might continue to have ringworm of the toes. but not become disabled.

It is of the utmost importance to grasp the interrelationship between these two very common skin conditions. There may be a coexistent occupational dermatitis and a ringworm infection. Either one may precede the other and lay a foundation for the other. The establishment of casual relation and the precise role played by occupation in these cases presents peculiar difficulties—not adequately disposed of by merely demonstrating the presence of the ringworm fungus between the toes.

Recourse to the patch test for the purpose of investigating the susceptibility of the individual to the chemical substance to which he was exposed still remains one of our most valuable aids to the diagnosis of occupational dermatitis provided one uses the method intelligently and with full cognizance of its limitations. It should never be omitted in doubtful cases, whether the ringworm fungus can be demonstrated or not. It is well to remember, however, that the clinical test—i.e., the fact that the condition clears up when exposure to the suspected irritant

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ceases, and recurs in re-exposure—is of the greatest significance even when the patch test turns out to be negative.

As in all other phases of medical diagnosis every available diagnostic procedure should be resorted to and the whole picture carefully appraised. Only then may one hope to arrive at a sound diagnosis upon the basis of which causal relation may be determined.

The Injection Treatment of Hernia

THE injection treatment of hernia, like the injection treatment of varicose veins, dates back more than 100 years.* At the outset the solutions used were too caustic. They stimulated the exudative phase of inflammation more than the proliferative phase. The modern solutions are milder and they stimulate more the proliferative phase and less the exudative phase. Another cause of failure in the early days of this treatment was the poor support given the hernias, bandages being used instead of trusses.

In 1835, Dr. Velpeau of France, originated this treatment by injecting a solution of iodine. Dr. Heaton and Dr. Pancost were his earliest followers in the United States. Dr. Billroth of Vienna, in 1880, recognized the possibilities of this method. He stated that if some mild solution that would cause proliferation of tissue could be discovered, hernia could be cured without a radical operation.

About 1890, the use of paraffin in the injection method of treating hernias was introduced. Paraffin acted as a moveable foreign body plug instead of an agent which could be absorbed, leaving much new scar tissue. The presence of this foreign body caused a slough, which necessitated its surgical removal. About this same time (1890), the theory of asepsis was definitely accepted, and Bassini, in Italy, and Halstead in this country, popularized the open method of operation. The success of the operative method and the miserable failure of the paraffin method caused the injection treatment to fall into ill repute. It lay dormant for about 20 years, except in the hands of irregular physicians.

During the last 30 years, more satisfactory solutions have been developed and trusses have been used to greater advantage. From this period, Dr. Pina Mestra of Barcelona, Spain, and Dr. Mayer of Detroit, have reported results which are very striking. Each of them claims several thousand cases treated with 98% cures. With such a high per cent of cures, I should imagine they treated only individuals with small hernias.

During the last five or 10 years the injection treatment of hernia has made its most rapid advancement and has finally been put on a scientific basis by Drs. Bratrud, McKinney, Bell and Rice of Minneapolis and others in New York. They made histological examination of tissues that had been injected with certain solutions, and demonstrated active proliferation of tissue without necrosis.

Any hernia that can be reduced completely and maintained in a state of reduction while the patient is ambulatory can be treated by the injection method. Strangulated or incarcerated hernias require surgery. Sliding hernias are difficult to hold by a truss and should rarely be injected. Postoperative or incisional hernias are usually large and contain adhered omentum or intestines with no definite sac; only the smallest ones should be treated by this method. Inguinal hernia associated with undescended testicle should be treated surgically. Any general surgical contra-indication, such as hyperthyroidism and hemophilia, should be considered a contra-indication, although poor surgical risks tolerate this method of treatment much better than surgery. Coughs should be be controlled before starting treatment; also straining from urethral stricture and prostatic hypertrophy should be controlled.

Any truss that holds the hernia completely reduced under all conditions will suffice. It is necessary to produce firm pressure over the hernial canal and for this reason the spring truss is usually the best type, however the elastic type may prove more satisfactory in individuals with rounded hips. The fastidious patient may require a rubber-covered truss, to permit his taking tub baths instead of sponge baths. If a patient is not accustomed to wearing a truss, the pressure should be gradually increased, and the truss worn day and night for a week before the injections are started. He must be impressed with the fact that the truss is not to be removed either day or night during the first two months, at which time the treatment is given. By the third month he may remove his truss at night, while in bed. The truss must be worn from four to six months longer, depending on the strength of the new wall.

TECHNIQUE: Indirect Inguinal Hernia—The patient is placed on the table, preferably in a slight Trendelenburg position. The truss is removed, and the skin over the hernial canal is "sterlized" with alcohol, or some mild antiseptic. A 5cc. syringe with a 24 gauge needle about 1.5 inches long is used. After making certain that the hernia is completely

* PENN RIDDLE, M.D., F.A.C.S., Dallas, Texas; read before the Section on Surgery, State Medical Association of Texas, Houston, May 28, 1936; published in *Texas State J. Med.*, July, 1936.

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reduced the needle is introduced through the skin in the region of the internal inguinal ring. It passes easily through the subcutaneous fat down to the facia of the external oblique muscle where it meets definite resistance. At this point, the operator should stop and renew the pressure gently so as not to pass the needle too deeply when he thrusts it through the fascia. The scrotum may be invaginated and the index finger inserted into the external, inguinal ring. This will act as a guide in estimating the exact area where the solution is to be injected. All injections should be made posterior to the fascia of the external oblique, never in the subcutaneous fat. The sac may be ignored. It will be compressed and obliterated by the newly formed tissue. The plunger of the syringe is slightly withdrawn and if blood is not aspirated, the solution is slowly injected; if blood is withdrawn, the needle is removed and reinserted. Also, if severe pain is experiencd, the needle is withdrawn and reinserted; the point may have been in contact with a nerve or within the abdominal cavity; in either event severe pain may result and the injection should cease. One or 2 cc. of Sylasol or Proliferol are injected at each treatment. If Proliferol is used, 2 cc. of 2% novocain must be injected, five minutes previous to the injection of the sclerosing solution. The treatments may be given from one to three times a week, depending on the reaction. About 12 to 16 treatments are necessary to close the average hernia. A fewer number of injections may suffice, but overtreatment is preferable to undertreatment. If too large a dose is given (about 3 to 5 cc.), there is apt to be swelling of the cord, more pain and a slight limp of a few days duration. It is better to give many small injections than a few larger ones. The first four or five injections are placed in the region of the internal inguinal ring, the remainder along the hernial canal, on either side of the cord and posterior to the cord, in the conjoined tendon and around the external inguinal ring. The truss is replaced before the patient rises from the treatment table. After about ten treatments are given, it will be noted that the hernial canal is full of fibrous tissue, and that there is no impulse on coughing.

Direct Inguinal Hernia—The point of attack is largely in Hesselbach's triangle, around the external inguinal ring. Much of the solution should be deposited posterior to the cord in the plane of the fascia of the transversalis muscle, and in the conjoined tendon.

Femoral Hernia—The solution is deposited around the femoral canal in a manner similar to the method of closing the femoral canal surgically. Injections are made into the same structures that sutures pass through—the inguinal ligament, Gimbernat's ligament, pectineus fascia and the femoral sheath, care being taken not to penetrate the femoral vein or artery. The relation of the femoral canal to the circulation in this region is, canal, vein, artery and nerve, from the symphysis pubis laterally.

Umbilical Hernia—A cone-shaped pad is used for support. Upon its removal, the injections are made in or near the fascia line, on all sides of the hernia. The index finger, inserted into the hernia by invaginating the skin, will act as a guide in placing the point of the needle. About six to 10 small injections (1 cc.) usually suffice.

Minor complications, such as swelling of the cord or penis, hydrocele of the cord and severe pain may occur if the treatment is pushed too vigorously (too frequent and too large doses), or if injection is made into the abdominal cavity. Rest in bed one or two days with hot applications will cause the condition to subside rapidly. Major complications, such as epididymitis, abscess, peritonitis, sloughs, sepsis, sterility and impotence have been mentioned, but with proper management these do not occur. The physician who is familiar with the surgical anatomy, pathology and diagnosis of hernia, and adheres to details of technique, need have no fear.

I have recently had the pleasure of visiting a number of the leading hernia clinics of this country and have received opinions on the injection treatment from the following surgeons: Dr. Bratrud of the University of Minnesota, Dr. Rice of the Minneapolis General Hospital, Dr. Smith of the Mayo Clinic, Dr. McMillan of Northwestern University and Dr. Girard of the Southern Pacific Hospital, San Francisco. All of these surgeons have been injecting hernias for from two to five years. Based on their opinions and my own short experience of one and one-half years, I believe the injection treatment will show 80% cure, where all hernias that can be held with a truss are treated. If the more difficult cases are eliminated, such as patients with large scrotal hernias (over 3 cm. in diameter) and those with flabby, fat abdomens and thin abdominal muscles, 98% can be cured by the injection method. This treatment will partially close any of the more difficult cases, and if surgery is later resorted to, the surgeon will have more tissue to work with. The difficult cases for injection treatment are also difficult cases for surgery. If surgery is attempted in the difficult group, a high per cent of failures will likewise result unless a plastic operation is performed, using fascia-lata sutures (autogenous). I tell my pa-

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tients that I will operate on them free, or charge them the difference in the two fees, if the injection treatment fails. In that event, I think only a plastic operation, using a fascia-lata suture, should be done. Putting the two methods on an equal basis (ignoring the fascia-lata plastic operation and removal of one testicle) I believe the injection treatment will cure as many cases as surgery. Most patients will not submit to a plastic operation or removal of the cord and testicle, until surgery has already failed once. Many cases in which recent surgery has apparently failed, can be treated successfully by applying a truss and giving a few injections. Occasionally complete surgical failures of long standing can be successfully treated by injection. Even the difficult cases can be improved to a state where a truss will hold back the hernia with ease. Many old individuals and others in whom surgery is contra-indicated (bronchiectasis, asthma, cardiorenal disease, hay fever and coronary disease) may be made more comfortable, if not cured. In 707 hernias treated, Dr. Bratrud reports "there has not been a single recurrence after the injection treatment that even approaches the size of the original hernia." Such results cannot be claimed for surgery where many patients have been made worse. In these 707 hernias, he found 17% were recurrences following surgery. Doubtless good surgeons can improve on this percentage, by obtaining permission from the patient to remove one testicle and the cord, or do a plastic fascia lata operation.

Recurrences following surgery or in the injection method may be due to the patient's inability to throw out fibrous tissue (fibroplastic diathesis[1]). We see very few hernias or hemorrhoids in the negro. This may be explained on the grounds that negroes throw out fibrous tissue (keloid tendency) very readily on stimulation by trauma or inflammation. One solution may stimulate fibrotic changes in one individual more readily than in another. For this reason, if a patient is not very responsive to one solution, it may be necessary to change to another. Sylasol[2] and Proliferol[3] are the most popular solutions manufactured today. I have used both of them with good results. I prefer sylasol as a routine, because it is mild, does not require an anesthetic, and is a very satisfactory proliferating agent. Where one solution has apparently failed, I try a few injections of another before recommending a plastic operation.

The advantages of the injection treatment have been partially outlined and are very obvious: ambulation, low cost, high safety factor, good results and no major complications.

The disadvantages are, irritation from wearing a truss, long duration of treatment and more work on the part of the surgeon. If a surgeon is not willing to learn to fit and adjust trusses, and to look after minute details of technique, he had better continue the operative method.

The injection treatment of hernia in the last one hundred years has risen and fallen in the estimation of prominent surgeons. Its development in the last 30 years has brought it to a stage where I believe it will prove as popular and effective as the injection treatment of varicose veins. The marked advancement in the last five years has been largely due to the fact that those who were well trained in surgical anatomy, pathology and diagnosis, have been working on the problem in a scientific manner and not by the "secret formula" method.

Abstract of Discussion:

DR. RUSSELL M. BELLAMY, Pampa: I believe that hernia as a structural defect can best be corrected by a surgical repair with living fascia. However, the fact remains that only a small percentage of hernias have in the past received this ideal type of treatment. Thousands of hernia sufferers are going through life wearing a truss with no intention or hope of being cured by a surgical procedure.

Industrial insurance with the accompanying employment examinations has placed a new economic importance on hernia and potential hernia. Fully 8% of all the men who report to us for examination have either a fully developed or a potential hernia. This disqualifies them for employment. Naturally, the disappointed applicant appeals to the examiner for a solution of his problem. Heretofore we have had to inform him that a major surgical operation with an appreciable outlay of money, together with about three months off duty, would be necessary to correct his defect. To many of these men this has been an insurmountable obstacle, so they have had to change the nature of their employment or work for smaller companies or contractors who did not require employment examinations. To this class of patients

1. ROSSER, CURTICE: Rectal Pathology in Negro; Incidence and Peculiarities, *J.A.M.A.*, 84: 93-97, January, 1925.

2. G. D. Searle and Co., Chicago.
3. Ulmer Pharmacal Co., Minneapolis.

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the injection treatment of hernia has been welcome news.

Although I would rather repair a hernia surgically, I believe that the injection treatment is entitled to a place in medicine. It is physiologically sound; it has been subjected to a thorough clinical trial, and the results are satisfactory. There is an important difference between the operative and injection treatment which should be clearly recognized from the beginning—that the injection treatment requires more complete cooperation of the patient. If the doctor or the patient is unwilling to follow the painstaking method necessary for a cure by this method, then it is better that the patient be subjected to a herniotomy from the start.

A doctor who is not qualified to do a surgical repair of a hernia is also unqualified to give a satisfactory injection treatment. A thorough konwledge of the anatomy of the parts is essential.

Rheumatic Fever—Occupational Disease?

RHEUMATIC fever is increasing in the United States. * The past winter and spring evidenced a decided increase in the incidence of the disease in Wisconsin.

A recent report of the American Committee for the Control of Rheumatism[1] states that studies indicate that the incidence of rheumatic fever depends on various factors of climate and environment, the patient's geographic habitat, his social condition, and seasonal change. The results of the Medical Research Council's investigation, which included familial incidence and heredity, familial evidence of sore throats, maternal care, exposure, sleeping conditions, clothing, birthplace, occupation, types and sites of houses, and so forth, was largely negative. It is, however, the belief of many that some of these factors are impartant; namely, that the incidence of rheumatic fever and rheumatic heart disease is low in the tropics and higher in the temperate zone. It is also high among persons living near the sea level or in basements, particularly in cold, damp months.

The American Committee states that the great majority of writers favor one of the variants of the bacterial theory: (1) the theory of bacteremia; (2) the theory of bacterial toxemia; (3) the theory of bacterial allergy. For each group some hold responsible one bacterium or its toxin or its antigen; others consider that any one of a number of bacteria or their toxins or their antigens may be causal. The fact that sore throat so often precedes the initial or recurrent rheumatic attack and that the disease, always one of low infectivity, occasionally assumes epidemic proportions, strengthens the bacterial theory.

Five types of microorganisms have been isolated from the blood of rheumatic patients: (1) a "sporulent" bacillus, anaerobic but facultatively aerobic; (2) a diplococcus, aerobic but facultatively anaerobic; (3) a streptococcus generally hemolytic; (4) diphtheroid organisms (aerobic coccobacilli); (5) enterococci.

The authors of the "Rheumatism Review" feel definitely that the cause of the disease has not been established. The fact that so many different organisms have been isolated and are considered the cause of rhumatic fever, suggests that probably no one of those so far isolated is responsible. Rheumatic fever is a fairly distinct disease entity. It would indeed be surprising were it found to be caused by many different organisms. Rheumatic fever uncomplicated by carditis results in a period of disability lasting several months. When rheumatic heart disease occurs such a period is greatly prolonged and occasionally permanent and total disability results.

Although the disease is most common in children, sufficient instances are found among adults, especially males, to bring up the question, at times, of industrial liability. This has occurred several times; in each instance it was claimed that the occupational exposure caused the disease.

Rheumatic fever occurs in all walks

* Editorial by H.E.M., in *Wisconsin M. J.*, October, 1936.

1. "The Present Status of the Problem of Rheumatism and Arthritis; Review of American and English Literature of 1934," *Ann. Int. Med.*, 9:883, January, 1936.

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of life, in all vocations, and in all types of industrial environment. Although there may be an occasional occurrence in one whose work necessitates exposure to cold and dampness, it has been the opinion of those involved in the industrial aspects of the disease that there is not sufficient evidence at present to warrant including rheumatic fever in the list of occupational diseases.

In any disease where the resulting disability is greatly prolonged or even permanent, the proof of industrial hazard should be very definite.

Integrated Control of Occupational Diseases

SURELY no proof is need for the establishment of the socio-economic importance of occupational diseases, at least for those who have been reliably informed of our recent experiences.*

It is also axiomatic that control and prevention are far superior to cure, viewed from any of the aspects concerning occupational disease problems.

Others have covered the medical and engineering control of occupational diseases. What I shall attempt to do, therefore, will be to bring together and integrate these methods of control—mention some of the technical difficulties which have arisen, consider practical applications of methods in the different groups interested, speak briefly concerning the evolution of control, and, finally, attempt to come to some rational conclusions and recomendations.

In considering the engineering control methods, we are concerned, (A) with the basic principle of first investigating occupational conditions; (B) measuring the severity of the hazard scientifically, then assessing the hazard; and (C) instituting the indicated measures which have been evolved. These control methods include: (1) a physical and hygienic survey of plant conditions; (2) occupational history and analysis of employees; (3) adequate information concerning the materials and processes used: (4) atmospheric and gross sampling of materials associated with quantitative physical and chemical determinations; (5) the rating of the hazard by comparing the findings in a given instance with accepted criteria; (6) an assessment of the efficiency of protective apparatus and devises; and (7) an adequate and proper interpretation of this information, so that it can be practically applied to the industrial problem involved.

If the above procedures give information which indicates the necessity of using various methods of protection, the following have been found useful in different combinations: (1) the use of mechanical devices, such as exhaust ventilating systems, respirators, masks, or helmets; (2) the changing of processes; (3) the substitution of nontoxic materials for those of proved txicity; (4) educational programs; and (5) the continued maintenance and checking of the efficiency of protective equipment. It is not claimed that the list is exhaustive; there may be other measures of equal importance, but it is believed that the principal ones have been mentioned, and it is also known that they have been used successfully.

The medical methods of control are likewise well known, but will be mentioned briefly: (1) pre-employment physical examinations, with which is associated a thorough occupational history going back if possible for a period of 10 years; (2) periodic examinations of employees in so far as this is possible, but especially in instances where known hazards exist; (3) laboratory examinations, both in preemployment and periodic examinations, to obtain evidence supplementary to physical examinations, and to determine whether the applicant or employee shows signs of the effects of occupational exposure; (4) educational programs, constantly acquainting employee and employer with the need for the use of medical control procedures; and finally (5) continuous and careful medical supervision, involving the principles of employee-placement and adjustment, as well as continued study of environmental conditions and the proper functioning of protective mechanisms.

* C. O. SAPPINGTON, M.D., DR.P.H., F.A.P.H.A., Consulting Industrial Hygienist, Chicago; read before the Industrial Hygiene Section of the American Public Health Association, at the Sixty-fourth Annual Meeting, Milwaukee, Wis, October 9, 1935; published in *American Journal of Public Health*, Vol. 26, No. 8, August, 1936.

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It will be seen that the object of engineering measures, simply stated, is to estimate the severity and nature of the hazard presented and to recommend the use of methods in the industrial environment to control such hazard, while the object of medical proceedures is to determine the nature and extent of the effects of the environment on the bodies of the exposed employees and, similarly, to recommend and institute measures for control and prevention.

Although it will not be possible to discuss all the technical difficulties, emphasis will be placed upon the lack of proper coördination or integration of engineering and medical methods of control and the results thereof. It often happens, for instance, that the assessment of hazards in industrial environment is interpreted entirely separately from medical findings on employees in that environment. Contrariwise, it more frequently occurs that the medical data constitute the sole evidence upon which judgment is made as to the existence of potential occupational disease hazards.

The fallacy here is evident, because occupational disease is the direct result of exposure to deleterious conditions, materials, or processes found in an industrial environment. There is, therefore, a distinct cause-and-effect relationship, which cannot be efficiently used unless the interpretation of this cause-and-effect relationship is made by a thoroughly qualified person who understands the evaluation of an occupational disease hazard in terms of its effects upon the human body.

We cannot expect that the engineer will be able to do diagnostic work—not should he be expected to understand the application of the fundamental principles of industrial toxicology— although in some instances he may be able to tell what will probably take place under certain conditions; likewise, in most instances, the industrial physician, who has had little training in or contact with the engineering principles of industrial hygiene, will not fully appreciate the importance of the environment as an etiological factor in the production of occupational diseases.

We thus have a condition in which the engineer is interested in his side of the work, chiefly the industrial environment, and the physician, largely with diagnostic problems. The result has been a lack of real coördination and integration of casual factors with physiological functions of exposed employees.

This lack of integration has been because of the scarcity of what we might call "qualified interpreters" who understand and appreciate the principles involved on both sides of this question and are able to make interpretations in simple terms, so that this combined information may be issued to, and used by manufacturers, insurance companies, attorneys, and others to whom it is of vital concern.

The physician-industrial hygienist is probably the one specialized type of person who can adequately take the responsibility for this situation. Unfortunately, few of these have been developed, and in order to make usable the information gained by application of the methods of control of occupational diseases, we cannot depend entirely upon those few who have fortunately been able to secure training both in the fundamentals of medicine and industrial hygiene.

It is obvious that we cannot expect engineers to become physicians; it does seem reasonable, however, to expect the industrial physician to superimpose upon his medical background and experience at least an appreciation and working knowledge of the principles of industrial hygiene to the end that he may be in a position to integrate intelligently the etiologic and pathologic factors concerned in occupational diseases. Certainly, the majority of occupational disease problems which come up for final decision are medical in aspect, and it seems rational to say that the physician must, in the final analysis, take the responsibility for these decisions. Examples of the smooth functioning of such a principle are to be found in the work of the Office of Industrial Hygiene and Sanitation, U. S. Public Health Service, and the Bureau of Occupational Diseases, State Department of Health of Connecticut, where the technical and administrative policies of the group are under the guidance and control of an experienced medical officer.

As Glenn Frank has so well stated: "The future of America is in the hands of two men—the investigator and the interpreter. We shall never lack for the administrator, the third man needed to complete this trinity of social servants. And we have an ample supply of investigators, but there is a shortage of readable and responsible interpreters, men who can effectively play mediator between specialist and layman. The practical value of every social invention or material discovery depends upon its being adequately interpreted to the masses. Science owes its effective ministry as much to the interpretative mind as to the creative mind. The knowledge of mankind is advanced by the investigator, but the investigator is not always the best interpreter of his discovery. Rarely, in fact, do the genius for exploration and the genius for exposition meet in the same mind . . . . The investigator advances knowledge. The interpreter advances progress. History affords abundant evidence that civilization has advanced in direct ratio to the efficiency with which the thought of the thinkers has been translated into the language of the workers."

It has seemed to me that the regular routine physical examination must undergo considerable adjustment before it can be properly applied to occupational disease potentialities, both in the routine examination before employment and during employment on hazardous jobs. The physician surely should have in mind those conditions and defects which may become aggravated by the specific exposure which the employee meets. The physical examination should always be interpreted in the light of the possible effects of industrial environment. In other words, the physician should take into account particularly the nature and severity of the hazard presented, in evaluating the defects or abnormal conditions found on physical examination. This would appear to be a more intelligent application of the industrial physical examination than has been widely and generally made.

To apply these principles successfully, the examining physician should be able to give an intelligent answer to the following questions: (1) Do you know the nature and severity of the occupational hazard into which the examinee is going? (2) Are you satisfied that the examinee does not possess any abnormal conditions or defects which might be aggravated by occupational exposure? (3) Do you know the nature of the protective devices which are provided for the use of the employee and whether such devices are known to function properly?

Through intelligent interpretative comments in the answer to these questions, the physician can serve industry must more efficiently than he usually has done in the past.

In applying the principles of coordination and integration to the more efficient use of information regarding the control of occupational diseases, we are confronted with the problems of manufacturers, insurance companies, attorneys, and industrial commissions. We cannot entirely separate the problems of legal and compensation aspects from those of prevention and control, for we learn preventive methods largely through the so-called "trial-and-error" system. What we know about preventive medicine, for instance, was largely gained through repeated and collected experiences concerning disease manifestations and the ways in which they have been contracted. This principle applies no less to the prevention and control of occupational diseases, and it is necessary that preventive methods be developed through continuous observation and study of the avenues through which they develop; in other words, in our time at least, it will be impossible to divorce the study of the occurrence and effects of occupational diseases from that of their prevention, if advancement is to be accomplished.

It means practically nothing to the manufacturer to tell him that he has air in which there are 50 million dust particles per cubic foot; that the free silica content of this dust amounts to 35 per cent; and that a particle-distribution study shows the majority of the particles to be below 3 u in greatest diameter. Nor does it do him much good to find out that he has, in certain processes, a lead-in-air content of 10 mg. per 10 cu. m. of air, or a concentration of 1,500 p.p.m. of carbon monoxide. What the manufacturer wishes to know is the significance of these figures and what they mean in terms of possible disability among his employees. Someone must explain these things to him promptly and plainly.

Insurance company representatives, including engineers, claim men and underwriters, ask the same questions. The engineers want to know just what exposure would be safe. The underwriting group wants to know whether Plant A, as surveyed, is a good risk to put upon the books.

Attorneys and members of industrial commissions desire to know whether a given set of industrial

conditions is an actual occupational disease hazard, whether the employee actually has the occupational disease in question, and whether such disease was contracted in the plant where the employee recently worked.

All of these are definite, practical problems regarding the etiology of occupational disease and its manifestations. If we can so assemble our information that we come to reliable conclusions and make these available, industrial medicine and hygiene will have performed a signal social and scientific service.

Clearly, the rational basis for intelligent underwriting and compensation for occupational diseases lies in obtaining over a period of many years, adequate and continued records of industrial exposure and its effects in producing disease and disability. This may take considerable time, effort, and patience.

It is my belief that we are in an evolutionary period regarding occupational diseases. If we look back on our experience with fires, we find a prototype of this evolutionary process. In the beginning, there was no fire insurance and great losses were suffered. Eventually a system was provided by which one could insure against fire and the losses resulting. People discovered that in many cases it was not possible to recover the total loss suffered. After years of this experience, someone developed the idea that fire prevention was less costly than expecting to recover losses through insurance, although it was advisable both to carry coverage and prevent fires. Today we have developed many methods for the prevention of fire, as well as having improved in our ways of fighting fires. We can draw a similar analogy between the evolutionary processes of occupational diseases and those of accidental injuries. Time was (and probably still is, in some quarters) when those in authority believed that the effects of accidental injuries may be fully covered by insurance. This conception has gradually given way to the principle of accident prevention, which, while not fully developed, has gone a long way.

We shall probably have to learn the same lessons in occupational diseases that have previously been taught to us regarding fires and accidental injuries. Prevention is by far the most economical procedure, viewed from any angle.

We may conclude that there is great need for more extensive use of the principle of integration in associating information and coming to more reliable conclusions regarding the causes and effects of occupational disease—such results to be applied in their prevention and control.

Such action would involve the training and employment of greater numbers of those whom we have been pleased to call "qualified interpreters," or, a combination of the following: (1) the use of consultants and advisers by manufacturers in working out their problems of occupational disease prevention and control; (2) adaption of available personnel to the work of insurance companies, in respect to industrial investigations, claims problems, and the underwriting of future risks; (3) the development of more scientific medico legal principles in the handling of legal and compensation problems; (4) the further enlightenment and training of engineers, physicians, insurance company representatives, attorneys, industrialists, industrial commissions, and associatees, through periodic conferences, meetings, and lectures.

If I have appeared to be presumptuous and have trod upon traditions and disturbed old trains of thought, we can take courage from the words of Henry Thomas Buckle:

"For the great enemy of knowledge is not error, but inertness. All that we want is discussion and then we are sure to do well no matter what our blunders may be. One error conflicts with another, each destroys its opponents, and truth is evolved. This is the course of the human mind, and it is from this point of view that the authors of new ideas the proposers of new contrivances, and, the originators of new heresies, are the benefactors of their species. Whether they are right or wrong is the least part of the question. They tend to excite the mind; they open up new faculties; they stimulate us to fresh inquiry; they place old subjects under new aspects; they disturb the public sloth; and they interrupt rudely, but with most salutary effects, that love of routine which, by inducing men to go groveling on in the ways of their ancestors, stands in the path of every improvement, as a constant, and outlying, and too often, a fatal obstacle."

Progress in Silicosis Prevention

INTERRUPTED by vacation periods, the meetings of the Advisory Committee on Dust Control Rules and Regulations for the Rock Drilling Industries will be resumed. * During the past few weeks the two subcommittees appointed at the July meeting of the Advisory Committee have completed their work.

These sub-committees are :

On definition of "injurious silica dust concentration" (i. e. the number of particles of silica per cubic foot of air): Dr. W. J. McConnell, Director, Industrial Health Section of the Metropolitan Life Insurance Company; Dr. Leonard Greenburg, Executive Director, Division of Industrial Hygiene, State Department of Labor; Dr. R. R. Sayers, United States Public Health Service; Dr. Leroy U. Gardner, Director, Saranac Laboratory for the Study of Tuberculosis.

On schedule of tests for approval of dust control equipment and methods in the rock drilling industries: George P. Keogh, Industrial Code Referee, State Department of Labor; Drs. Greenburg and Sayers, and Daniel Harrington, Chief of the Health and Safety Branch, United States Bureau of Mines.

At the July meeting of the Advisory Committee the general scope and purpose of the rules and regulations to be recommended were tentatively agreed upon and definitions tentatively accepted except the one referred to the first of the above subcommittees and also the term "silica bearing rock" or "siliceous rock" which was reserved for further consideration.

It should be pointed out that this Advisory Committee is considering only the rock drilling industry as affected by Chapters 887—888 and 889 of the Laws of 1936. Industries other than rock drilling in which a hazard is created by harmful dusts are being considered also, and rules and regulations therefor will be drafted by committees. The schedule of tests for approval of dust control equipment and methods which has been prepared by the second sub-committee, does not apply to equipment and methods used for the removal of ordinary dust and fumes and for ventilation of factories. These latter equipment and methods, which have been in use heretofore with the approval of the State Department of Labor, are still necessary and are still approved. The 1936 legislation provides that "the Industrial Commissioner and the Industrial Board are hereby required to add to the Industrial Code * * * effective rules and regulations governing the installation, maintenance and effective operation in all industries and operations WHEREIN SILICA DUST OF OTHER HARMFUL DUST HAZARD IS PRESENT, of approved devices designed to eliminate such harmful dusts and to promulgate such other regulations as will effectively control the incidence of silicosis and similar diseases." Emphasis on this distinction is occasioned by inquiries which have come to the Labor Department which indicate that some factory operators believed they could not install blowers or suction systems for the removal of ordinary dust and fumes until such equipment had been approved under the tests to be recommended by the Advisory Committee on equipment for the rock industries.

Because of the high cost of workmen's compensation insurance for silicosis due to lack of definite and reliable information non silica dust hazards and their effects in New York State industries, Industrial Commissioner Elmer F. Andrews in collaboration with Dr. Thomas Parran, Jr., then Commissioner of Health, and Superintendent of Insurance Louis H. Pink, some months ago inaugurated a silicosis survey in the foundry industry to obtain full and exact facts as to working conditions and the physical effects upon the workers therein.

Dr. Greenburg summarizes the progress of this work as follows:

"A study of silicosis in the Foundry Industry has been in progress for some time. All of the foundries in the State of New York known to the Department of Labor have been surveyed. X-ray pictures and occupational histories have been taken of approximately 4,300 workers in foundries. When this work is completed, 5,000 such workers will have been thus studied. The x-rays in hand have already been interpreted by two experts and are now in the hands of a third. It is expected that this field work on the foundry silicosis study will be completed within the next month."

THIS CONSTITUTES THE LARGEST FOUNDRY SILICOSIS STUDY EVER MADE ANYWHERE IN THE WORLD and should be of great value not only in New York but in other states.

Other operations within the State

* *The Industrial Bulletin*, State of New York Department of Labor, September, 1936.

Department of Labor under the new laws have progressed apace.

Under the requirements of Chapter 887 there is being prepared a list of physicians from which the Special Medical Examiners provided for in the Chapter will be appointed. Under the same Chapter, two Expert Consultants on dust diseases, Drs. J. Burns Amberson and Edgar Mayer, have been selected, and selection of the third is under way.

Theodore Hatch of the Harvard School of Public Health, one of the outstanding experts on methods of dust sampling, analysis and control, has been added to the personnel of the Division provisionally as Associate Dust Control Engineer. Dr. Leonard Goldwater, of New York University Medical School, specialist in industrial hygiene and occupational diseases, has been added provisionally as Senior Industrial Hygiene Physician to specialize in research on silicosis and other dust diseases.

Industrial Hygiene Exhibit

THE Division of Industrial Hygiene this year stressed silicosis in its exhibit at the State Fair, held at Syracuse, September 5-12.*

The material shown was chosen with a view to presenting simply and clearly the salient points about the disease. Thus, there were posters showing in what industries silicosis most commonly occurs, and under what circumstances it may be expected, i. e., where the quartz content of the dust is high, dust exposure continuous, etc. Other posters showed the amount of dust by actual dust count in familiar industries and the quartz content of familiar dusts.

There were micro-photographs of various dusts and a collection of dust samples labelled as to their silicosis producing properties.

The stages of silicosis were demonstrated both by x-ray prints and transparencies. A poster stating that tuberculosis follows silicosis, brought home the point by charts illustrating the amount of tuberculosis among the granite cutters of Vermont compared with the amount among adult males in the general population. Another x-ray print showing a case of tuberculo-silicosis accompanied the poster and a second set of transparencies showed damage from dust in the case of an asbestos worker, a rock driller, a granite cutter and a foundryman.

Two lung specimens showed the actual appearance of a lung affected by silica and one affected by coal dust.

Under the heading "Silicosis is Incurable. Prevent it. Control the Dust", a series of photographs showing practical methods of dust control in rock drilling, granite cutting and other industries was displayed. Dust control in metal grinding was illustrated by an ingenious model.

Finally that part of the exhibit dealing with dust was completed by the demonstration of two types of positive air pressure helmets for use in supplying the worker with pure air from an outside source when the situation is such that the dust cannot be effectively removed.

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* *The Industrial Bulletin*, State of New York Department of Labor, September, 1936.

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